

The four intrahepatic structures and their interrelationships (Dorsal view)
blue portal vein red hepatic artery yellow bile ducts gray hepatic veins



INTRODUCTION TO HEPATIC SURGERY

by

HENRY GANS, M D

SURGICAL RESIDENT CINCINNATI OHIO

with an Introduction by

A BRUNSCHWIG M D

ATTENDING SURGEON TO THE MEMORIAL HOSPITAL
FOR CANCER AND ALLIED DISEASES NEW YORK N Y
PROFESSOR OF CLINICAL SURGERY CORNELL UNIVERSITY
MEDICAL COLLEGE NEW YORK N Y



ELSEVIER PUBLISHING COMPANY

AMSTERDAM HOUTON LONDON NEWYORK

1955

ALL RIGHTS RESERVED THIS BOOK OR ANY PART THEREOF MAY
NOT BE REPRODUCED IN ANY FORM (INCLUDING PHOTOSTATIC
OR MICROFILM FORM) WITHOUT WRITTEN PERMISSION FROM THE
PUBLISHERS

PRINTED IN THE NETHERLANDS BY M. V. DRUKKERIJ C. J. THIJM NIJMEGEN

*Dedicated to the Memory of my Mother
and to my Father*

PREFACE

Comme la chirurgie des membres celles des viscères doit être régie par l'anatomie de la région sur laquelle on opère et aussi par la connaissance des lésions contre lesquelles on intervient

La chirurgie viscérale doit tendre à être une chirurgie réglée comme l'est depuis longtemps la chirurgie des membres Il est donc nécessaire de préciser au point d' vue chirurgical tout ce qui touche à l'anatomie des régions sur lesquelles on opère

HARTMANN 1901

A case of sickness in our own immediate surroundings confronted us with the fact that in certain cases of pathological affections many surgeons are deterred by the very great risks involved from resecting parts of the liver even though they feel this to be necessary.

The operation techniques followed have certainly helped to cause an ambivalent attitude in the surgeon. The methods introduced round about 1880 by Von Bergmann, Langebuch, Von Eiselberg, Keen and others are difficult and at the same time so primitive that serious complications threaten the patient both during and after the operation.

For this reason, almost every successful partial liver resection was indeed still is, made public, from which it is obvious how hazardous the surgeons themselves considered this intervention and how relieved they were that their action was not followed by death. (Bax and Schalm 1954)

Unaware at first of a recent analogous research by Counaud we attempted to avoid the defects of the old method by taking new paths. From the beginning we kept two criteria in mind in the first place the method to be developed must be anatomically justified which could by no means be said of the old method and in the second place it must be simple. No method can be introduced into general surgical practice unless it can

be executed in most simple means which every operator has at hand' (Garre, 1907)

In order to meet the first criterion, we investigated the anatomy of the various intrahepatic structures. The gaps in the knowledge of the course and interrelationship of the various intrahepatic structures, which we noticed in consulting the anatomy text-books, have long been recognized, though perhaps from another viewpoint than ours. As an illustration of this may serve the observation by Mann, in 1942, that the history of the blood circulation in the liver lies buried beneath its own literature. Thus, for example, in 1898 Cantlie in a short communication entitled "On a new arrangement of the right and left lobes of the liver", showed that "the present anatomical division (of the liver) into right and left lobes is unscientific and consequently untrue and untenable", whilst one still finds the liver described in current anatomy books as being built up of two lobes.

In the last years, however, a gathering interest in the anatomical structure of the liver has become apparent. A number of studies have appeared which deal with the structure of the lobules (Ehasz, 1953, Rappaport *et al*, 1954) as well as of the further anatomical structures distinguishable in the liver (Hjortsjo, 1948, Ellis *et al*, 1952, Herley *et al*, 1953, Couinaud, 1952 and later). In the work of Couinaud the segmental construction is given great prominence. We shall also devote a large part of our study to this problem, also in connection with the surgery of the liver.

Of importance in surgery are some long forgotten publications by Wendel and Martens (1920), in which a description is given of an attempt to approach hepatic surgery in a manner that is anatomically justified. They not only set the ligation at the liver hilum of the structures of that part of the liver to be resected as the crucial point of their method, but they also pointed out the importance of a certain anatomical plane in the liver along which the resection should be made.

Another explanation of the tendency to solve the problems with the help of anatomy is found in the spectacular way in which lung surgery has profited from the study of the anatomy of the intrapulmonary structures. Thanks to the application of plastics to the already old injection corrosion technique not only has the anatomical investigation of the various structures, and especially of their spatial relationship, become much simpler, but it has also produced completely new views, so much so indeed that we feel, as a result of our research, that a deepened knowledge of the anatomy of the liver not only helps to solve the problems of hepatic surgery, but also furnishes a possible basis for entirely new operative possibilities.

Nijmegen May, 1955

HENRY GANS, M D

ACKNOWLEDGMENTS

From this place I wish in particular to record my debt to the Members of the Surgical Staff of the Jewish Hospital, Cincinnati Ohio who guided my first steps in surgery, and who granted me a leave of absence to finish this work. To my former chief Dr A GROLLMAN, I would express especial thanks not only for his teaching and encouragement but also for his friendship.

During the time that I was assistant in pathology in the St Canisius Hospital, Nymegen the director of the Department of Pathology, Dr J G PLETTE kindly made it possible for me to examine livers by the corrosion technique, and set time at my disposal for studying the literature. For this and for the interest he showed I am very grateful.

Dr H J LAMMERS, professor in anatomy at the Medical School of the R C University Nymegen gave me the opportunity to go over the material scientifically, without his greatly appreciated help and valuable criticism this work would never have developed into a dissertation.

Dr H R BAX chief surgeon at the Municipal Hospital Arnhem, gave me the chance on several occasions to assist during operations on patients with symptoms of liver tumour or carcinoma of the gall bladder, and further I was able to operate on a few dogs in his clinic. His interest and moral support were of considerable help to me and I am very grateful to him.

I should like to thank Dr J H J M PERNET surgeon at St Canisius Hospital for enabling me to put the acquired anatomical insight into practice on one of his patients.

I received a great deal of help from Dr H J G WIJERS, professor in pathology, Medical School, R C University, Nymegen, in working on the difficult subject of surgical hepatic pathology. I am greatly indebted to him for his interest and his aid in solving the different problems.

For reading the typescript of the chapter on pre- and post operative care and for their valuable criticism I am indebted to Dr L SCHALM, medical director, Municipal Hospital, Arnhem, and Dr C A A HAANEN, medical resident, St Canisius Hospital, Nymegen.

I acknowledge with pleasure the efforts of the draughtsman and the photographer of the Medical School, R C University, Nymegen, who were required to work to such meticulous detail for the accomplishment of this work. They are Mr C VAN HUIJZEN, who did the drawings and who sketched the various intrahepatic structures from a great number of corrosion preparations, this being of great help in assembling Chapter IV, and Mr A REIJNEN who took care of the photography. I should also like to thank Mr F COENEN, of the St Canisius Hospital for his assistance.

To Mrs A CALON SNEEVLIET I owe a special debt of gratitude for her enthusiastic help in typing the manuscript.

To Miss M HOLLANDER I am greatly indebted for her valuable help with part of the translation and the correction of the manuscript.

To all not explicitly mentioned, who have contributed towards the completion of this work, I take this opportunity of expressing my thanks.

H G

CONTENTS

INTRODUCTION		xv
CHAPTER I	On the subdivision of the liver	i
CHAPTER II	Embryology of the liver	40
CHAPTER III	The Glissonian system	49
CHAPTER IV	Variations in the structures of the Glissonian system	64
CHAPTER V	The hepatic vein system and its varia- tions	92
CHAPTER VI	Survey of the subdivision of the liver	108
CHAPTER VII	Indications for hepatic surgery	117
CHAPTER VIII	Introduction to hepatic surgery	152
CHAPTER IX	Surgical approach to the liver	183
CHAPTER X	On hemi hepatectomy and some hepato lobectomies	188
CHAPTER XI	The care of the patient before, during and after hepatic surgery	199
APPENDIX I	Employed injection corrosion tech- nique	219
APPENDIX II	Hepatography	224
Summary		235
Literature		238
Author Index		257
Subject Index		261

INTRODUCTION

Hepatic surgery is not new. There are literally hundreds of individual case reports and reports of small groups of operations upon the liver for various neoplasms and cysts, both benign and malignant. Also in recent years attention has been focused upon the development of portal shunting procedures, envisaging the relief of ascites in the presence of cirrhosis. Moreover aneurysms of hepatoportal vessels seem to be receiving more attention both as to clinical diagnosis and operative obliteration.

Rational surgical procedures presuppose a detailed knowledge on the part of the surgeon of the anatomy of the organ and tissues in question. The rather complex anatomy of the liver has been described in the past and the field again studied recently. The facts as brought out by these older and more recent studies are certainly not appreciated by the majority of abdominal surgeons. These points as well as many others are discussed at length by Dr. Gans in this book. Hemorrhage, which is the principal immediate complication in excisional hepatic surgery, will be rendered less likely when surgeons plan their operations, taking into consideration the anatomical principles so well described and emphasized here.

In the general expansion of surgery interest in operations upon the liver is being revived, judging from the recent literature. Unlike the past when no one surgeon had appreciable personal experience in operations upon the liver, it may be predicted that such experience will increase.

Dr. Gans' treatise is thus very timely. He emphasizes the broad aspects of fundamental knowledge especially anatomy

that surgeons should appreciate in this type of work. This volume should be read and digested by those anticipating operations upon the liver. In addition, it should stimulate surgeons to think more about the liver as an organ that can be safely operated upon when the indications exist and not, as in the past, a field that would seem beyond surgical attack, or if attacked, a field highly fraught with danger.

ALEXANDER BRUNSCHWIG, M.D.

New York City, N.Y., August 15, 1955

Chapter I

ON THE SUBDIVISION OF THE LIVER

The subdivision of the liver both in its external aspect and on the basis of the internal arrangement of the intralobular hollow structures has been the subject of profound study all down the ages

In this first chapter we shall follow the gradual unfolding of knowledge in the past on the external division and shall then proceed to discuss the internal division of the organ as described formerly and now. Finally we shall see how this external subdivided pattern is related to the internal one

A THE EXTERNAL SUBDIVISION OF THE LIVER

The study of the anatomy of the liver is as old as culture itself. It appears from Sueda's (1900) excellent paper on some ancient illustrations of human livers found by archaeologists that one of these objects—a small clay model of a sheep's liver—dates from the Babylonian era; its age has been estimated at approximately 4–5000 years (fig. 1).

The priests of those days were guided during religious ceremonies and when consulted by the observation of variations encountered in the entrails of animals whereby each variation observed had a specific meaning.

Haruspicina was therefore not solely restricted to magic practices but included a science based upon a study of the

different forms and variations of the intestinal organs. Stedius included these models in the curriculum for sacerdotal students

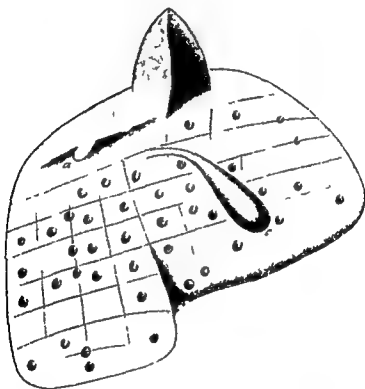


Fig. 1 Baked clay model of a sheep's liver
(The liver tablet present in the British Museum) (Stedius 1900 by courtesy of
Bergmann Verlag München)

Each lobe of the sheep's liver, the so called *penula* or *penicula* also named *fibra* or *augmentum hepatis* had its own significance, which appeared from the names *mensa* *focus* *cultus* and *aurum*, by which each individual lobe could be distinguished from the others.

Galen's conception, probably derived from his observations of animals, assumed that the human liver was also a lobated organ, an opinion which was corrected by Vesalius: "Quare enim dissectionem professores de jecoris formam ac penulis seu fibris (qua lobis Græci vocant) commentantur e canum

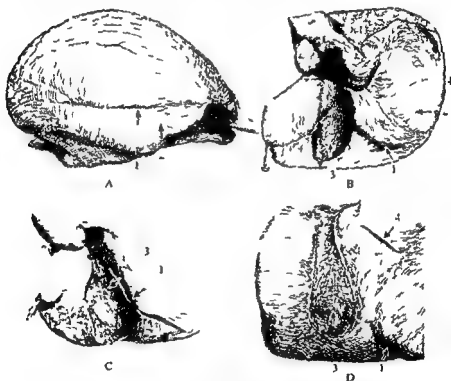


Fig 2 A liver from right lateral B C and D liver seen from behind
1 fissura interlobaris dextra 2 lobus dexter 3 fossa cystica 4 incisura dextra
(after Ruge 1911)

potius et seminarum sectionibus, quas hominum didicerunt — Humanum enim jecur porcini et multo adhuc minus canini jecoris modo, non discinditur Nam unicum continuumque præcipue ipsius substantia corpus hominum jecor est

("The professors preferred to describe the external form of the hepatic penulae or fibræ (which the Greeks called lobæ))

with the help of dissections on dogs and monkeys than to teach with the help of dissections on man

For the human liver has not as in the pig and still less as in the dog, a subdivision into lobes, the liver is a unity, and principally by its own substance one cohesive whole')

In most of the present day textbooks of anatomy one seldom encounters any other subdivision of the human liver than into right and left lobes. The site of this division is marked by the incisura umbilicalis at its ventro caudal margin, the insertion of ligamentum falciforme at the ventral and cranial convexity and the fossa umbilicalis et ductus venosus (Arantii) at the dorso caudal or visceral liver concavity

However, other divisional patterns have been described in the past, the first systematic description of the lobation which can be distinguished at the dorsal aspect of the human liver being by Von Haller (1764), who in his turn derived a great deal of his knowledge from earlier anatomical studies such as those of Vesalius, Eustachius and Spiegel

Von Haller's conception must be considered in some detail here, seeing that, on the whole, very little of this important work has been preserved as common knowledge

This author recognized two fossae sagittales at the dorsal liver surface united by a transverse one. The latter is commonly indicated as 'sulcus transversus' or 'porta hepatis', which name is derived from the word *πύλη* used by Aristotle (fig 25)

The fossa sagittalis sinistra corresponds with the fossa venae umbilicalis and fossa ductus venosi (Arantii), the fossa sagittalis dextra corresponds with the fossa vesicae felleae on its caudal and the fossa venae cavae on the dorso cranial side

In this way Von Haller distinguished four liver lobes, divided by three fossae namely the lobus dexter, located to the right of the fossa sagittalis dextra, the lobus sinister, located to the left of the fossa sagittalis sinistra, and the lobus medius located between the right and left fossae sagittales subdivided by the fossa transversa into an anterior part or lobus anterior

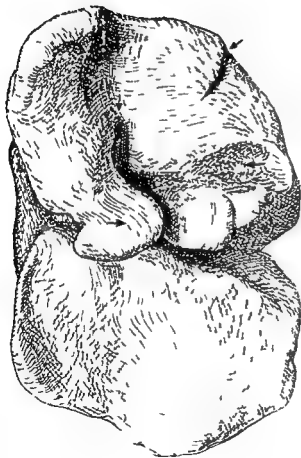


Fig 3 Liver dorsal aspect
 1 inferior margin 2 fissure in inferior margin (according to Ruge) 3 fovea cystica 4 lobus caudatus (From Ruge 1911)

and a posterior projecting one, the lobus posterior, which Von Haller named later on lobus caudatus, bordered to the right by the inferior vena cava, to the left by the ligamentum venosum (Arantii), ventrally and caudally by the fossa transversa and dorsally by the crura dorsali liver margin. The lobus caudatus was subdivided by Von Haller into two colliculi, the one located alongside the fossa sagittalis dextra, the colliculus caudatus, the other along the fossa ductus venosi, directed caudally, the colliculus papillaris divided from each other by a shallow fissure. Later the term colliculus was superseded by 'processus', but many names have been used other than these in the course of time.

We shall therefore first deal briefly with the nomenclature which has been used in the past to indicate different parts of the liver.

Sylvius (1478) designated the lobus caudatus as lobus minimus. Minimus ad portam exortum, Vesalius as tuber 'quod duodenum exporrigitur et vena portae e jecore prodit jecur quodam tubere prominet' and Spiegel (1622) as lobus exiguus. lobus exiguus בלתי אטומים nondum descriptus. Von Haller introduced the name lobus posterior, later terming it the lobus caudatus, and its subdivision into the two aforementioned colliculi, Rex (1888) introduced the name lobus omentalidis Ruge (1909), lobus venae cavae, subdivided into a lobus dorsalis and a lobus descendens.

The lobus quadratus has also been denoted by various names, i.e., lobus interior (Von Haller 1764), lobus quadrangulus (Muschka, 1863), lobe principale moyen (Cuvier & Duvernoy, 1835) central suspensory lobe (Rolleston, 1861), lobus portaalis (Ruge, 1909) and many other names.

For this reason the adoption of an agreed uniform terminology was felt to be necessary. Accordingly since the introduction of the B.N.A. terminology (1895), 'lobus quadratus' (Spiegel) is the accepted term for that part of the liver which

is located between the porta hepatis, ductus venosus and inferior vena cava and which is limited towards the right by an impression, corresponding with the insertion of the hepato duodenal ligament

'Lobus quadratus' (B N A terminology) stands for the part of the liver which is located between the fossa umbilicalis fossa cystica and sulcus transversus portarum, while by lobus dexter is understood the part to the right of and by lobus sinister the part to the left of the insertion of the falciform ligament

The differences in the external aspect of the various types of livers encountered in the different mammals had so puzzled many investigators that eventually *comparative anatomical* studies of the organ were undertaken. The purpose of all these studies was to find one pattern of division to which all liver types both the lobated and the different unlobated varieties could be reduced

For the lobated liver as it occurs in pig dog and many other mammals shows a number of fissures radiating from the inferior or as the case may be ventro caudal liver margin towards the centre of the liver mass, dividing the organ into a number of lobes the number varying in the different mammalian species. To realize such a pattern Ruge and De Burlet independently searched in a great number of human livers for rudimentary or fully developed fissures corresponding to those which are generally considered to be the main fissures of the lobated liver

Between 1909 and 1919 Ruge as well as De Burlet described a number of human livers showing fissures which answered their purpose (figs 11 and 81 p 96). These fissures one in the left liver lobe linke Seitenspalte (fissura sagittalis sinistra) and one in the right liver lobe rechte Seitenspalte (fissura sagittalis dextra) were the two new fissures which they added to the ones previously described by Von Haller namely the incisura umbilicalis and fossa cystica. In this way both the lobat

ed and the unlobated liver can now be subdivided into 2 lobus lateralis sinister, a lobus centralis, composed of three 'Stamm lappen' (Ruge and De Burlet), namely the lobus centralis sinister the lobus præportalıs and the lobus dextro vesicalıs, and the lobus lateralis dexter, located to the right of the rechte Seitenspalte¹

The lobus dorsalis, corresponding with the lobus caudatus was seen to be subdivided by 1 fissure into 1 lobus venæ cavae turned to the right, and 1 lobus descendens towards the left lateral lobe

In all investigations dealing with this subject (Duvernoy Rolleston, Flower, Cantlie, Girard, etc.) the main body of the mammalian liver is divided either into two halves each of which is subdivided into a number of lobes (Flower, Cantlie, and others), or into three main parts, viz the right, middle (or central) and left lobe (Ruge and others), the middle lobe being subdivided into either two or three lobes (table 1)

We shall revert to this controversy later (Chapter VI)

II THE INTERNAL SUBDIVISION OF THE LIVER

From Stieda's study it becomes obvious that the Haruspices not only observed the liver surface but also the liver substance by means of incisions, the holes which can be seen in one of the liver models, the cellie of the Roman priests represent the open lumen of veins which, as we know, do not collapse but expand when this organ is incised¹

One of the first and most comprehensive investigations of the anatomy of the human liver and its intrahepatic structures is laid down in the study 'Anatomia hepatis' by Glisson (1654). In this work the author deals extensively with the intrahepatic course of both portal and hepatic veins and their interrelationship. On account of the fact that Glisson was able to clarify the relations of the different structures to each other with the aid of some fine illustrations his work rose

TABLE I

		Left lobe	Left princip	Principal lobe	Right princip	Right lobe
CADDER DUVERNOY	1835			Middle princip		
				Suspensory lobe		Right lobe
RILLESTON	1861	Left lobe	Left suspens lobule	Central suspens lobule	Right suspens lobule	Right lobe (superior lobe)
OWEN	1863	Left lobe		Cystic (gall) lobe		Right lobe
FLOWERS	187	Left lateral lobe	Left segment Left central lobe		Right segment Right central lobe	Right lateral lobe
CANTLIE	1898		Left side			Right side
BERGÉ	1901			Central lobe		Right lobe
GIRARD	1830	Left lobe				Right outer lobe
GIBALT	1850	Left outer lobe	Left inner lobe	Right inner lobe		Right lobe
LENGING AND MÜLLER	1873	Left lobe	Left Central princip lobule	Central lobule	Right lobule	Right lobe
LEIBENBERGER AND BAUM	1891	Left principal lobule (lobus sinister)	Left central lobule	Central principal lobe (lobus medius)	Right central lobule	Right principal lobule (lobus dexter)
RICE AND DE BRUIT	1902 1911	Left lateral lobule (Lobus sinister)	Left segment (lobus sinister)	Right segment Preapical lobe (lobus apicalis)	Left lateral lobule (lobus dexter)	Right lateral lobule (lobus dexter)
RFX	1888	Left inferior lobe	Left superior lobe	Central lobe		Right superior lobe
MEYER FRIED	1911	Left lateral lobe	Left central lobule	Infraapical central segment	Right central lobule	Right lateral lobule
CHARNOCK BRADLEY	1929	Left lateral lobe	Left lobule			Right lateral lobe (lobus dexter)

above the mere descriptive representation of data and therefore fully deserves to be counted as the starting point of the many following anatomical studies on the liver

There is no explanation for the remarkable fact that Glisson's name only has been preserved in connection with the

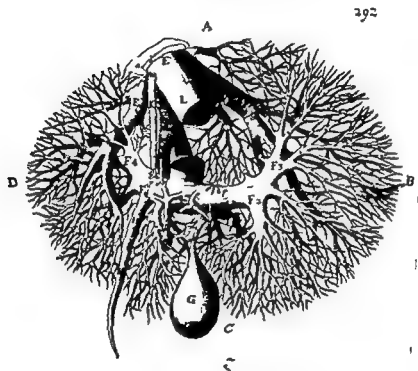


Fig 42 Relation of portal vein to hepatic vein seen dorsally (after Glisson (1631))

hepatic capsule whereas it is now a matter of common knowledge that this structure had been described previously by Waleus or Varolius (1640) the original description however, was ascribed to Jean Pecquet, a medical student who also gave a first account of the thoracic duct

The erroneous name "capsule of Glisson" was introduced by Francis Kiernan (1833) who dealing with the histological

liver unit the lobulus, described the portal triads as 'containing the hepatic ducts, the portal veins, the hepatic arteries, and the terminal branches of all these vessels with the nerves and absorbents enveloped in a sheath of cellular tissue, first described by Glisson and called Glisson's capsule

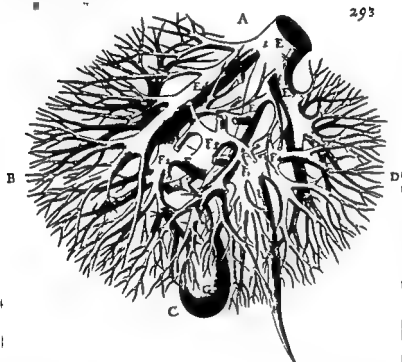


Fig 4b Ditto seen ventrally

From this quotation it appears that Kiernan especially pointed out that intrahepatically the bile duct arterial branch and portal vein branch run in close proximity to each other a common connective tissue sheath capsula Glissoni, envelops the corresponding structures. The three structures entering or leaving the liver at the hilum or porta hepatis are therefore regarded as forming one morphological system

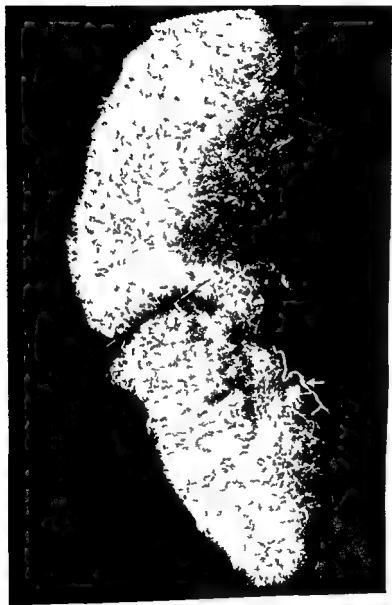


Fig. 6. Specimen of portal vein. Cranial aspect. The plane of the median fissure forms an angle of $60-75^\circ$ with the horizontal plane through the hilum; the aperture of the angle being turned in the 1 ft. direction. An arrow points to the intrahepatic portal vein and the portal vein.



Fig. 7. Specimen of portal vein.

* Sub-arterial vessels of the gall bladder divided into two halves by the medial fissure.

zontal plane through the hilum of about 60-75°, the opening of this angle is turned to the left (figs 5 and 6)

This fissure, the *fissura media*, is a constant one, showing very little variation from the course described above. It divides the gall bladder into two parts of nearly equal size, one to the left and one to the right.

When the portal vein is injected the subserosal venous capillaries of the gall bladder are filled even after previous ligation of the cystic vein. Numerous anastomoses proved to be present between the minor intra-hepatic portal branches and the subserosal vessels of the gall bladder (fig 7).

Both the quadrate and left lobes are situated to the left of this fissure thus they form the left part of the liver. Moreover, the *fissura media* divides the caudate lobe into two parts located on either side of the fissure. We possess several corrosion specimens of livers of which the right and left trunk of the hepatic arteries and/or bile ducts were alternately injected. The injection was preceded by ligation of the left or right branch. In none of our corrosion specimens could we find any anastomoses between the bile duct systems on both sides of the *fissura media*. This confirms the recent observations of Schalm (1952), who used Von Hildebrand's X-ray technique (see Appendix I) for his investigation.

Three groups of arterial anastomoses between the vascular beds of right and left hepatic artery have been described by Martens (1920) and Segall (1923), both using that same technique, which were also present in our specimens.

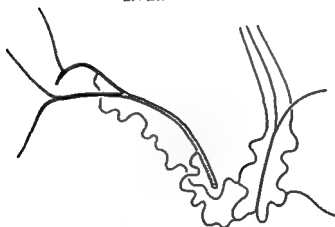
The first plexus is found extrahepatically in the hilar area (fig 8). It was present in about 25% of our specimens showing 15 fine branches arising from the right and left hepatic artery.

The second group of anastomosing vessels, which are branches of the fifth to the seventh order, were found in the area of the dorsal surface of the caudate lobe.

These vessels cross the fissura media but they will cause hardly any bleeding during hepatic surgery because of their small calibre



LIVER 60



LIVER 54

Fig II Arterial anastomoses in the h far a ea

A third plexus which was present in only a few of our specimens consisted of intra- and subcapsular single or multiple anastomosing usually very fine arterial branches which also may cross the fissura media. In only one specimen did this plexus consist of vessels with a fair calibre

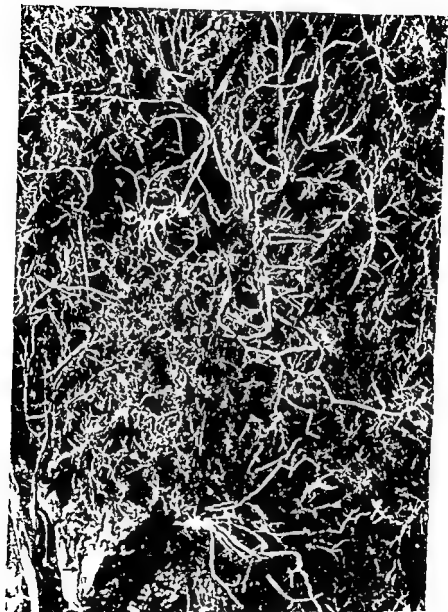


Fig 9 Subcapsular arterial anastomoses in the area of the caudate lobe
→ Portal vein

On the basis of these observations one may say that the liver is a paired organ for the arborization of its Glissonian structures. The left liver half, though usually not as large as the right liver half is considerably larger than the left liver lobe.



Fig 10 Microscopic sect on of the liver in the area of the fissura media
A fissura media B portal triads

Histological examination of the liver was performed in the region of the fissura media. The liver was cut perpendicular to the direction of this fissure. On microscopic examination it was found in a series of sections that the slides—which were smooth and did not show any further tears or cracks—displayed a fissure at the same level where we expected the fissura media to be present. This fissure as a tear formed a definite plane in the series of slices dividing the liver tissue in the centre of liver sinusoids. Small portal triads were observed on either side of the tear (fig 10).



Fig 11 Ventral view of a portal vein specimen 1 Median fissure 2 Left interlobar fissure
3 Right interlobar fissure 4 Intrahepatic fissure in right lobe

The gall bladder, as will be seen presently, develops out of the pars cystica located at the medial and caudal pole of the pars hepatica. During the development, this organ keeps its central position. It is therefore not surprising to see that the fissura media divides the fossa cystica into two parts of nearly equal size. Occasionally, however (8-10% of specimens) the fissura media may run along the left border of this fossa.

Two fissures can be distinguished in the liver halves described previously (fig. 11), dividing the Glissonian system of both halves into four parts, approximately corresponding to the liver lobes of the lobated liver, e.g. as in dog or pig (Rex, 1888, Hjortsjo, 1948, Healey *et al*, 1953-54, Couinaud, 1954, Gans 1954).

There is one frontal fissure to the right and one sagittal fissure to the left of the fissura media. On the left side is the *left interlobar fissure* corresponding with the external division between the left and right lobe. It is marked at the inferior liver margin by the incisura umbilicalis, the medio visceral part harbours the sagittal part of the left portal vein trunk (recessus umbilicalis of Rex, 1888), which is identical with the most cranial portion of the left umbilical vein. The cranio visceral part of this fissure corresponds with the fossa of the ductus venosi (Arantii) which duct after originating at the curvature of the left portal vein trunk, runs cranially towards the inferior vena cava. post nally it is obliterated. At the ventral surface it corresponds with a line slightly to the left of the insertion of the falciform ligament.

On the right side of the fissura media there is the *right interlobar fissure* the ventral surface of the liver usually shows nothing to suggest its presence. Its intrahepatic course shows some variations depending upon the divisional pattern of the right portal vein trunk (figs. 12, 13 and 14). Generally in the corrosion specimen this fissure cuts the lower margin of the ventral surface at a point between the fissura media and the right

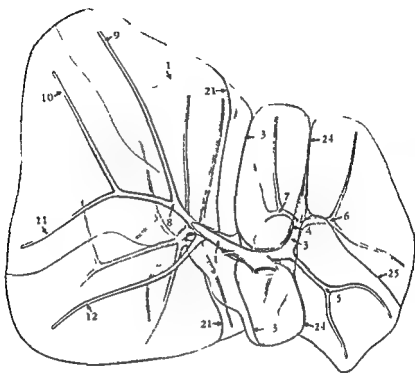


Fig 12a

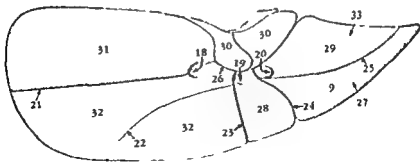


Fig 1 b

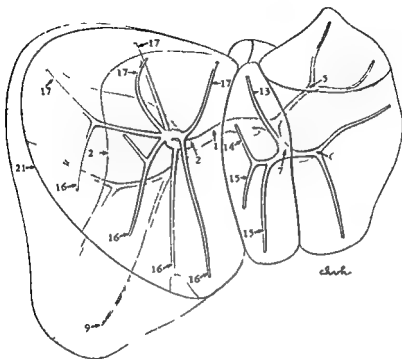


Fig 12c

Fig 12 Variations in the course of the portal vein I

A Dorsal aspect B Cranial aspect C Ventral aspect

- 1 Truncus dexter venae portae 2 vena paramedialis dextra 3 pars sagittalis venae portae sinistrae 4 saccus 5 vena cranialis lobis sinister 6 vena caudalis lobis sinister 7 vena paramedialis sinistra caudalis 8 vena dextra 9 vena paracystica 10 vena dextro caudalis 11 vena dextro cranialis 12 branch of the dorsal fan 13 vena paramedialis sinistra cranialis 14 branch of 7 to the ventro cranial area of the lobus paramedialis sinister 15 branch to the caudal area of the lobus paramedialis sinister 16 branches to the caudal area of the lobus paramedialis dexter 17 branches to the cranial area of the lobus paramedialis dexter 18 vena hepatica dextra 19 vena hepatica media 20 vena hepatica sinistra 21 fissura interlobaris dextra 22 fissura intermedia 23 fissura media 24 fissura interlobaris sinistra 25 fissura intersegmentalis sinistra 26 fissura dorsalis 27 segmentum ventro caudale lobis sinister 28 lobus paramedialis sinister 29 lobus sinister 30 lobus caudatus 31 lobus dexter 32 lobus paramedialis dexter 33 segmentum cranio-dorsale lobis sinister

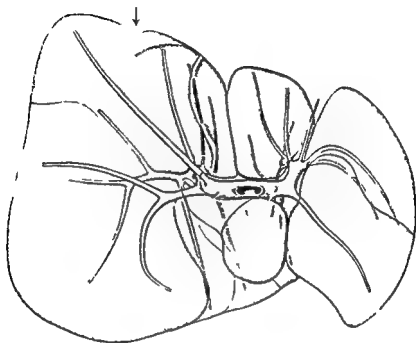


Fig 13a



Fig 13b

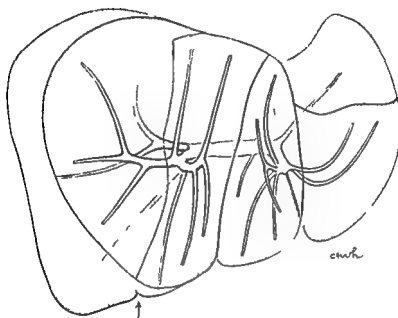


Fig 13c

Fig 13 Variations in the course of the portal vein II

A Dorsal aspect B Cran al aspect C Ventral aspect

→ An incisure which is present in the fresh liver does not correspond with the right interlobar fissure

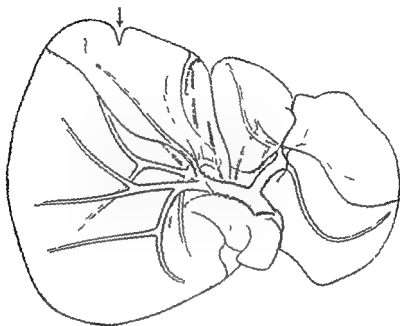


Fig 14a



Fig 14b

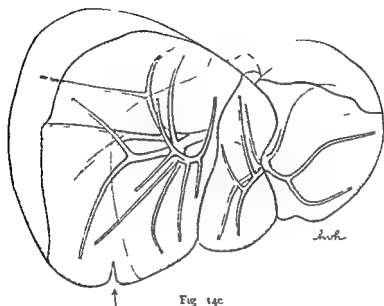


Fig 14c

Fig 14 Variations in the course of the portal vein III

A Dorsal aspect B Cranial aspect C. Ventral aspect

→ An incisure which is present in the fresh liver does not correspond with the right interlobar fissure

liver margin, situated at approximately one third of the distance from the latter. Over the ventral surface it runs upwards for a distance of 10–15 cm parallel to the right liver margin (depending upon the size of the organ) following which it curves to the left to terminate at the right side of the inferior vena cava at the point where the right hepatic vein enters this

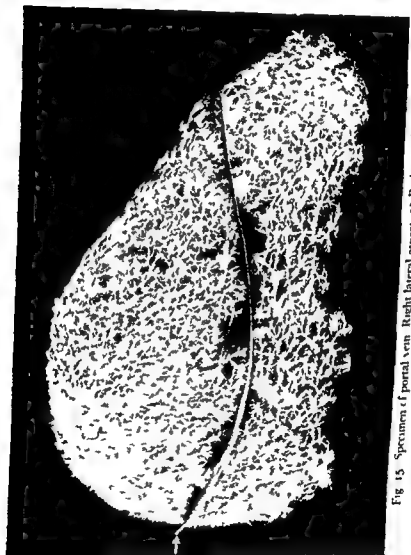


Fig 15 Specimen of portal vein Right lateral aspect → Right interlobar fissure

vessel. This plane cuts the dorsal surface of the liver along a fairly straight line, going through the basis of the right portal median vein. This oblique fissure makes an angle of 30-45° with the horizontal plane with the opening of the angle turned to the right (fig. 15).

In our polyvinylite specimens the three fissures described can easily be visualized by bending the casts slightly. In this way the Glissonian vessels can be distinguished without doing much harm to the normal anatomy.

We were able to inject some of the lobar portal vein branches individually with different coloured plastoid. A sharp demarcation of the lobar vessels appeared to exist, without any anastomoses of the portal vein beds of two adjacent lobes.

This fully agrees with the opinion voiced by Elias & Pettit (1952), that portal shunts normally are absent. It is probable that isolated anastomoses in a single specimen are the result of some local pathology, not detected at autopsy especially since current observations in these laboratories have shown numerous portal anastomoses in cirrhosis.

Small arterial anastomoses are occasionally found between the different lobes, usually subcapsular, seldom more centrally in location.

C. RELATIONSHIP BETWEEN THE EXTERNAL AND INTERNAL SUBDIVISION OF THE LIVER INTO LOBES

Is there any relationship between the external division of the organ (Ruge and De Burlet) and the internal one of the Glissonian system?

To answer this very important question for hepatic surgery we compared fresh livers with injection corrosion specimens.

Concerning the left interlobar fissure and the medial fissure we have already answered this question for the *fissura mediana* is located in the *fossa siggatahs dextra* described by Von Hal-

ler, which, as we have seen, is composed of the fossa cystica and fossa venae cavae, while the left interlobar fissure cor

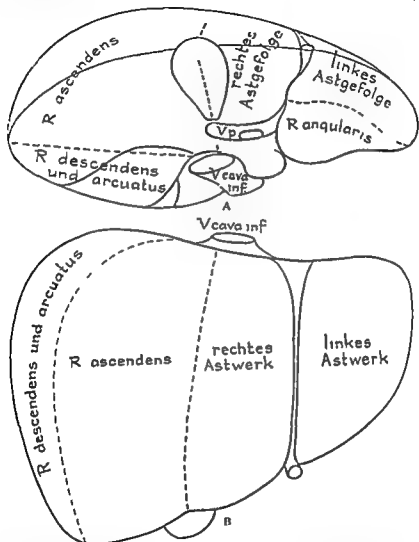


Fig 16 Division of the intrahepatic portal vein flow bed after Rex
A Caudal aspect B Ventral aspect (re-drawn after Rex)

responds with the fossa sigillaris sinistra of Von Haller and not with the linke Seitenspalte described by Ruge, which is located far more laterally

To investigate the right interlobar fissure we injected a number of livers showing incisure at the inferior margin of the right liver lobe, which according to Ruge, should correspond with the rechte Seitenspalte (fissura interlobaris dextra (fig 3 p 5) These two however certainly were not in

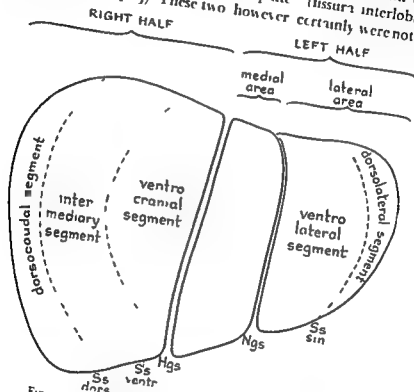


Fig 17 Division of the Glissonian system after Hjortso
Ventral aspect (re-drawn after Hjortso)

variably related as is clear from figs 13 and 14. The plane of the right interlobar fissure an area which has been shaded in these figures usually cuts the dorsal liver surface either along the right margin or along a line through the right wall of the cavity which corresponds with the fossa cystica. This deviates considerably from the line drawn by both Healey (1953) and Couinaud (1954) to mark off this fissure (fig 18-19).

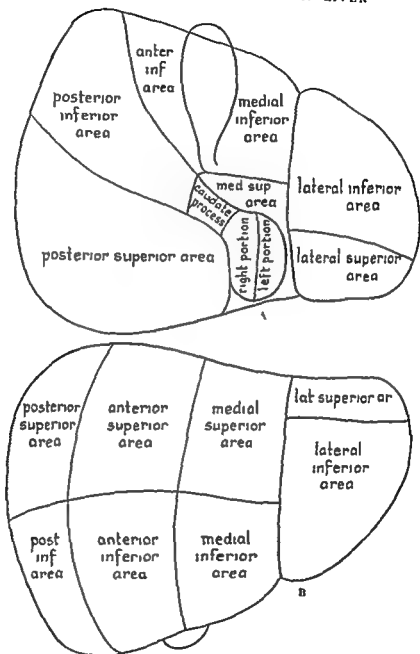


Fig 18 Division of intrahepatic bile-duct and hepatic arterial bed after Healey & Schroy

A Dorsal aspect B Ventral aspect (re-drawn after Healey & Schroy)

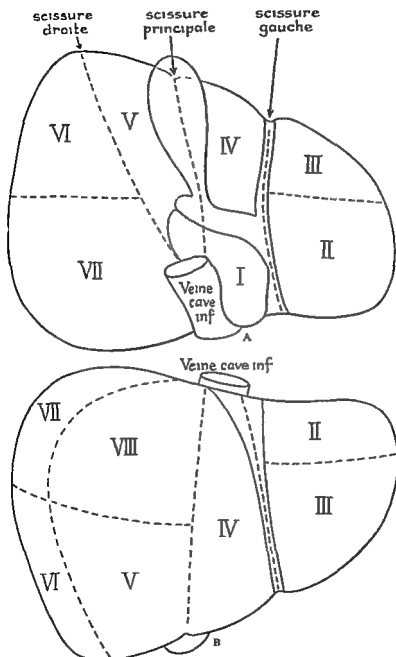


Fig 19 Division of the Glissonian system after Couinaud
 A Dorsal aspect B Ventral aspect (redrawn after Couinaud)

The incisuræ which were found in the fresh specimens are located at a good distance laterally from the point of intersection of this plane with the inferior liver margin.

In other instances, as in the livers reported by Ruge in 1911 (fig. 4), and in our specimen (fig. 20), a fissure was nevertheless found in the fossa cystica, which he described as follows:

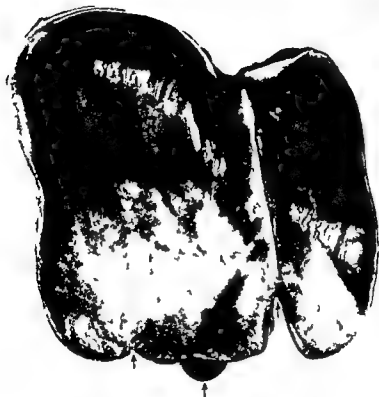
'Subseröse Gefässe liegen in der Furche die sich dorsally durch die Gallenblasegrube in oberflächlicher Lage verfolgen lassen, senken sich aber vor der Pforte in die Tiefe ein'

("In the fissure lie sub serosal vessels which can be followed dorsally through the gall bladder cavity in a superficial position, but they sink into the deeper part of the tissue before reaching the portal fissure")

In 10 % of our corrosion specimens Couinaud's line corresponded with the localization of the right interlobar fissure at the dorsal liver surface (fig. 14). In those instances we frequently found a shallow groove (sulcus) or a real fissure at this site in the fresh specimens, but the latter may also be present in those instances in which the fissure does *not* correspond with these grooves.

In view of the fact that Rex and Hjortsjo each invented different and rather intricate terms for the internal division of the organ, we aimed at adopting a *nomenclature* which would at the same time be consistent, anatomically, and easily understood and memorized. Therefore, we distinguished on the basis of the ramifications of the Glissonian system, a hepater dexter and a hepater sinister, divided from each other by the fissura media, for both lobes located on either side of this fissure we coined the term lobus paramedialis ventralis sinister, or dexter, as the case might be.

Both lobes lateral to the fissura interlobaris have been indicated as lobus dexter and lobus sinister, the lobus dexter to the right or laterally and dorsally to the fissura interlobaris dextra the lobus sinister to the left of the fissura interlobaris sinistra.



1

2

3

Fig 20 Liver ventro-cranial aspect
1 fissura interlobaris dextra 2 gall bladder 3 incisura umbilicalis

The lobus dexter, for the external division of the organ, does not correspond with the lobus dexter for the internal division, for which reason we shall henceforth designate the former as the *pars dextra (hepatis)*

The lobus caudatus, which is located between the right and left interlobar fissure, appears to be divided by the fissura media into two lobes lobus paramedialis dorsalis dexter and sinister. The two interlobar fissures do not approach each other intrahepatically in the median line, but intersect the dorsal surface of the liver on both sides of the lobus caudatus. Hence the *lobi paramediales dorsales* are situated between the right and left interlobar fissure. These small lobes are separated from the two lobes paramediales ventrales by a frontally placed fissure—called the *fissura dorsalis*—which cuts the cranial pole of the liver in an arc (fig. 12). Cranially, this fissure cuts the superior wall of the fossa transversa (or porta hepatis) in virtually a straight line.

It is currently known that the lungs can be seen to be divided into a number of lobes. On injection of the bronchi and pulmonary arteries followed by corrosion of the organ, it appeared that the external division into lobes corresponded with the internal arborization of the bronchi as well as the arteries but subdivision of the lobes into segments, not present on the outer part of the lung were also found in this way.

Does this likewise apply to the liver?

We find that the different lobes described previously for the Glissonian system, like those of the lungs similarly fall into a number of segments*. The term "segments" was probably introduced by Flowers (1872), though he used it in the reverse sense. Quand le foie est ainsi partage en deux masses principales, ne presentant pas de subdivisions, on peut les nommer

* The right word is actually sector because the liver area indicated by this term is three-dimensional. Our use of the less correct term segment is in deference to its general adoption in medical nomenclature & x in pulmonary anatomy and surgery.

lobe droit et lobe gauche, mais, lorsque ces deux masses sont divisées on doit les designer sous le nom de segments droit ou gauche du foie, en reservant le nom de lobe pour leurs subdivisions"

(Thus, when the liver is divided into two main parts showing no subdivisions one can call them the right lobe and the left lobe, but when these two parts are sub divided one must refer to them as the right and left segments of the liver reserving the name lobe for their sub divisions)

Rey (1888) did not speak of segments for the subdivisions which he found in his *portal vein* specimens He however distinguished two areas in the lobus dexter : Astgefølge des Ramus Ascendens and Astgefølge des Ramus Descendens und Arcuatus and two delimited areas in the left liver lobe

Linkes Astgefølge and Astgefølge des Ramus Angularis (fig 16)

Hjortsjo (1948) is alone (see fig 17) in stressing the fact that his S^s sin represents a sagittal fissure He did not note any of the transversal fissures described by Healey *et al* and Counaud (figs 18 and 19)

Counaud (1954) who distinguished eight segments indicated by numbers instead of names arrived at the division which does not correspond with his conception of the symmetrical arrangement of the intrahepatic Glissonian structures (see page 110) moreover his division differs considerably from Healey *et al*, (see figs 18 and 19) who came to a division into ten segments

It will be clear from this brief survey that there are some discrepancies between the results of the various recent findings and these call for further investigation Our own work however has facilitated the task of discovering exactly how these lobes were subdivided there are in fact several alternative means of establishing the segmental borders with absolute certainty namely

- (1) by bending the cysts apart. In this way the different segments stand out clearly,
- (2) by breaking off the segmental structures of the Glissonian system at their base,
- (3) by injecting the segmental structure of each individual segment with plastics of different colours.

Our specimens show that the left liver lobe is neither subdivided by a transversal nor a sagittal (Couinaud, Healey), but by a frontal plane, which takes a different course from that indicated by Hjortsjo (compare fig. 17 with fig. 12). This *fissura intersegmentalis lobii sinistri* divides the left liver lobe into a *segmentum cranio dorsale* and a *segmentum ventro caudale*.

As far as can be judged from Ruge's illustrations, the *fissura intersegmentalis lobii sinistri* seems to correspond with the one described by this author on the dorsal surface of one of his fresh livers, which he indicated as 'linke Seitenspalte', described as a 'Querspalte' (a transversal fissure!). Although we never encountered this fissure in our fresh specimens, a slight hint of it may occasionally be found on the dorsal left lobar surface as a shallow groove.

Unlike the left lobe, the *lobus dexter* is divided by a transversal fissure (see fig. 11) into a *segmentum craniale* and a *segmentum caudale*.

No clear cut transverse fissures are present in either paramedian lobe, this contrary to Healey and Couinaud.

A sagittal fissure, *fissura intermedia*, can be recognized, dividing the right paramedian lobe into a medial and lateral territory, to which we shall revert in Chapter III (page 59).

Conclusions. The liver can be divided both on its external aspect and on the basis of the arborization of structures of the Glissonian system.

In the external subdivision we distinguish a *pars dextra* and a *pars sinistra*, *lobus quadratus* and *lobus caudatus*.

For the internal division we were able to confirm the fact that the liver is a paired organ. Moreover, the intrahepatic Glissonian flow bed is divided into four ventral lobes viz the lobus dexter and sinister and both lobi pyramediales ventrales and two dorsal lobes, the lobi pyramediales dorsales which only partly correspond with the external division.

The grooves seen on the visceral surface do not always correspond with the fissures recognized for the internal subdivision.

Both the lobus dexter and lobus sinister are subdivided into two segments.

Chapter II

EMBRYOLOGY OF THE LIVER

HAVING considered the division of the liver on the basis of the arborization of the Glissonian system in the preceding chapter, we shall now briefly survey the most prominent features in the development of the human liver which are pertinent to the present and future interpretation of findings.

As is generally known, the *liver parenchyma* develops from the entoderm of the ventral wall of the fore gut. The first "Anlage of the liver parenchyma is paired in the turtle, some birds and reptiles (Ihle *et al.*) and also was thought initially to be paired in the human embryo (Hertwig, 1888), until Thompson (1908) found only one hepatic bud in a 2½ mm foetus.

This bud arises in a 2-2½ mm (12-17 somite stage) embryo, which is at an earlier stage than that at which the lungs and pancreas develop.

The hepatic cells invade the septum transversum, located between the pericardial cavity on the cranial and the yolk stalk on the caudal side. The invasion is accompanied by destruction of part of the mesodermal cells, which are the original constituents of the septum. Initially the cells lie embedded in between the septal mesoderm, but they quite soon increase in density on account of their rapid proliferation.

The liver cells are arranged in a trabecular spongework around sinusoids, lined with a layer of flattened mesothelial cells, through which the blood from the vitelline as well as the

umbilical veins is distributed. The rich blood supply of the organ accounts for its rapid enlargement, taking place at first in a lateral and dorsal direction, as right and left hepatic masses soon followed by that in a ventral and caudal direction. In the first stage of its development the enlargement of the liver is out of proportion to that of the rest of the foetal organism.

At a very early stage the hepatic bud divides into a cranial part which develops into the liver parenchyma (*pars hepatica*) and into a dorso caudal part, developing into the gall-bladder and cystic duct (*pars cystica*).

The *pars cystica* initially located intrahepatically (De Burlet, 1911) grows out as gall bladder and cystic duct. During the development of the liver the gall bladder probably occupies a central position. Die ursprüngliche Lage der Gallenblase entspricht der Medianebene, dass diese ursprüngliche Lage aber im Laufe der Entwicklung dieselbe bleibt ist nicht bewiesen, eine Verschiebung an der Leberintestinalfläche ist wegen der frühen, tiefen Einbettung im Parenchym nicht wahrscheinlich. (The original position of the gall bladder corresponds to the median plane, but there is no proof that this original position remains the same in the course of development. Owing to the early deep embedding in the parenchyma it is unlikely that there is any displacement with regard to the *facies intestinalis hepatis*.) (De Burlet)

The hollow stalk, connecting the *pars hepatica* with the duodenum differentiates into common bile and common hepatic duct as also into both hepatic ducts and their intra-hepatic ramifications.

The development of the *hepatic venous system* in connection with the subdivision found in the portal flow bed presented a problem. It appears that during the process of lateral extension of the liver the dorso lateral surfaces of both hepatic masses soon meet the vitelline veins running along the primitive alimentary tract which are broken up into sinusoids, invaded by the liver cells.

Extrahepatically each vein differentiates into a distinct distal* or afferent and a proximal* or efferent structure for the liver. We imagine, with Mall (1906), that at this moment two primary lobules come into existence, towards the end of the third week, each one with its own afferent and efferent vein. If this is what happens, then it is obvious that, though the parenchymatous "Anlage" of the liver is unpured, its internal arrangement is from the very beginning a purified one, around both vitelline veins.

The umbilical veins, which run in the lateral abdominal wall, communicate in the septum transversum with both efferent vitelline veins and discharge their blood by way of the truncus vitello umbilicalis into the sinus venosus.

Both lobules, extending into the septum, subsequently reach the umbilical veins, which in their turn are broken up into sinusoids. The ingrowing cells derived from both primitive lobules, should we imagine, form two new ones, again each with its own afferent and efferent vein. At this stage of four primary lobules corresponding with the primitive lobes, the sinusoids still consist of one wide system of channels which are in open communication with each other. The primary lobules are very small: at the end of the second month the diameter is 0.5 mm, at birth 1 mm and in the adult 1.5 mm (Mall, 1906).

In general, the same course of events takes place during the development of all mammalian livers. As far back as 1900 Choronshtitzky stated that the right half of the liver of a sheep embryo is supplied by the right vitelline as well as the right umbilical vein while the left side is supplied by both corresponding left veins. Charnock Bradley (1909) found in pigs the left and right umbilical veins to be located on either side of the gall bladder.

Several changes occur in the extrahepatic venous system, subsequently followed by intrahepatic rearrangements, during which the two intrahepatic flow beds, those of the porto-

* In relation to the heart

umbilical afferent and efferent hepatic vein system, are formed

The afferent or distal segments of the originally *paired* vitelline veins communicate by three cross anastomoses, the cranial and caudal ones being located ventrally and the medial one dorsally to the alimentary tract. Following obliteration of the right vitelline vein part, which is located in between the caudal and medial anastomoses and that part of the left vitelline vein in between the cranial and medial anastomoses, only the right vitelline vein is left over to transport the blood from the alimentary tract to the liver. This vein assumes a composite S shape which as superior mesenteric vein after rotation of the gut curves over the pars inferior duodeni and, as portal vein underneath the head of the pancreas and the pars superior duodeni (His, 1885)

Moreover, the extrahepatic distal part of the right umbilical vein is obliterated in the 6 mm embryo, the proximal part located in the septum transversum has been incorporated in the extending liver tissue as vena hepatica dextra located between the right and left paramedian lobulus

Von Haller (1774) while discussing the changes which occur at birth remarked that blood returning from the placenta passes into the liver through vessels that in the foetus are offshoots of the umbilical vein but in the adult are offshoots of the portal vein (Barclay *et al*, 1952). Probably the same thing happens during the early development of the organ when the right distal umbilical vein is obliterated

The four primitive lobules which are formed in this way are thought by us to correspond with the four ventral Glissonian lobes described previously. After the obliteration of the proximal left vitelline vein the blood of both paramedian lobes is discharged by way of the right proximal vitelline vein which shifts to a position in between both lobules its extrahepatic part the ductus hepato cardiacus becomes the proximal part of the inferior vena cava

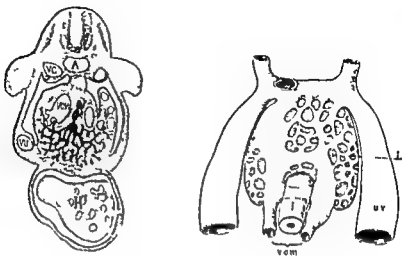


Fig 21a

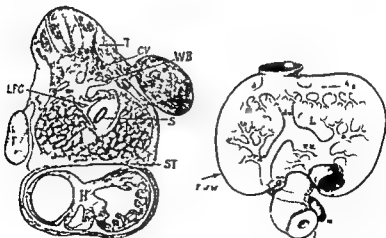


Fig 21b

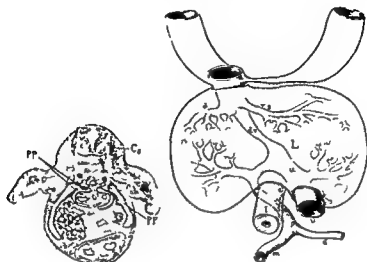


Fig 21c

Fig 21 Semi-diagrammatic reconstruction of the venous systems of the liver

A embryo of 4½ mm B embryo of 7 mm C embryo of 9 mm

l liver uv umbilical vein vom omphalo-mesenteric vein intestine c cardiac en s stomach lpc lesser peritoneal cavity h heart st septum transversum ru recessus umbilicalis pp pleuro-peritoneal cavity p vena portae dv ductus venosus rhd ramus hepaticus dexter rha ramus hepaticus sinister ra ramus angularis ra¹ ramus arcuatus (from original of figs 9 10 16 17 19 and 20 Franklin P Mall Am J Anat V 236 260 and 261)

This process of internal rearrangement starting at the end of the fifth week has been described by Mall (1906) in the following way. The hepatic and portal veins are telescoping they are beginning to dovetail with each other. The different stages of this process are represented in fig 21.

The four ventral lobes can be recognized as separated for a shorter or longer period in the embryo. De Burlet still found clearly marked fissures radiating from the margo inferior towards the centre of the liver mass in a 32 mm embryo. These fissures coincided with the medial and interlobar ones described for the adult human liver (fig 22).



Fig. 22 Model of a liver belonging to a human embryo of 32 mm (De Burlet 1911)

A Ventro-cranially B posterior aspect

- 1 Fissura interlobaris dextra 2 fissura media 3 fissura interlobaris sinistra
 4 lobus dexter 5 lobus paramedialis dexter 6 lobus paramedialis sinister
 7 lobus sinister 8 lobus caudatus

The lobuli paramediales dorsales (caudati) develop somewhat later than the four ventral lobules Mall described their formation thus With the subdivision of the middle and left hepatic veins into two branches each, six primary lobules are seen to correspond with the six primary lobes of the mammalian liver and 'The primary lobules radiate from a centre and have between them the main trunks of the portal veins, each interlobar vein at this stage is to form a main trunk in the adult

The development of the *hepatic artery* takes place after fusion of the most cranial ventral branches of the descending aorta into the coeliac artery One of its branches takes its course to the liver as hepatic artery

Many errors in the development of this artery account for the numerous variations encountered in the hepatic arterial blood supply (Rio Branco, 1912)

Initially the liver extends in a lateral direction then in a ventral and caudal direction the site at which both vitelline veins enter the liver the *hilum*, originally located near the lower liver margin being pushed up into a relatively more cranial direction Thus the portal vein enters the organ at its dorsal surface slightly cranially and dorsally to the bile ducts which have been differentiated from the original connective stalk between hepatic bud and gut

At the hilum of the adult human liver we still find traces of the original situation in the form of a figure H the two vertical bars of which represent the right and left vitelline veins while the horizontal bar corresponding with the sulcus transversus of Von Haller, represents the aforementioned cranial anastomosis between the two vitelline veins The right bar is identical with the main portal vein and right portal vein trunk The left vitelline vein corresponds with the medial portion the left umbilical vein with the lateral portion of the pars sagittalis venae portae sinistrae the left cranial bar with the ductus venosus (Arantii) (fig 23)

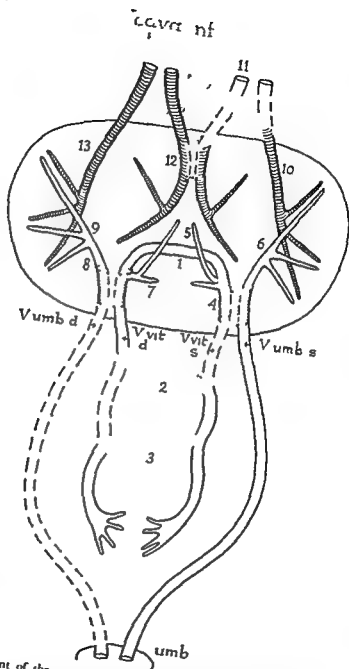


Fig 23 Development of the intrahepatic venous systems in diagram. Anastomoses between the venae omphalo-mesentericae 1 cranial anastomosis 2 medial anastomosis 3 caudal anastomosis 4 pars sagittalis 5 venae paramediales sinistae 6 vena cranialis and caudalis lobis sinistri 7 vena paramedialis dextra 8 vena dextra 9 vena cranialis and caudalis lobis dextri 10 vena hepatica sinistra 11 obliterated proximal part of the left satellite vein 12 vena hepatica media 13 vena hepatica dextra

Chapter III

THE GLISSONIAN SYSTEM

Prior to a systematic discussion of the structures comprising the Glissonian system extrahepatically in the hepato duodenal ligament and the hilum as well as intrahepatically, and of the relationship of the lobar and segmental structures to the hilum, the lobes and segments concerned we first have to define the conceptions 'Glisson's capsule' and 'hilum'.

Glisson's capsule The triangular, coronary and falciform ligaments the lesser omentum the hepatic capsule the hilar and the intrahepatic connective tissue, all blend without any transition because these structures are derived from the mesoderm of the septum transversum. The hepatic capsule named after Glisson, dives into the organ at the hilum as a sheath around the portal vein branch and its corresponding artery and bile duct for which reason all the hilar connective tissue has—in our opinion rather arbitrarily—likewise been considered as Glisson's capsule. In the dissecting room specimen represented by fig. 24 the two intrahepatic systems are readily distinguished but the three structures of which the Glissonian system is composed are so densely interwoven that it is exceedingly difficult to separate them.

Hilum The hilum is defined as the area at the dorsum of the liver where after removal of all loose connective tissue Glis-

sonian system structures can be recognized extrahepatically prior to their penetration into the body of the liver. It includes the sulcus transversus (Von Haller), the umbilical fossa and the incisura dextra, the latter extending from the right margin of the porta hepatis in a crudo lateral direction. This incisura

A



Fig. 24. Dissecting room specimen, dorsal aspect. Glissonian system. A at the hilum. B intrahepatic. C the medial hepatic vein.

is present in approximately 80 % of livers, its length varies from 3 to 5 cm. After drawing back the overhanging liver margins which partly cover the incisura, a rather shallow space appears which is a continuation of the porta hepatis. The hilar structures can be further dissected out at the bottom of this space.

The relationship between the different hilar structures

becomes clear during the surgical approach to this region (fig 25), with the ventral liver surface turned cranially and its inferior margin now directed upwards. In this way one faces the dorsal or visceral surface of the liver.

In this approach the most cranially situated structures—bile ducts and arteries—first become visible, while the most cranially located one—the portal vein—lies deep down, becoming visible only after previous dissection of both the bile ducts and the arteries.

After this brief description of the hilum, we must now dwell on the topography of the Glissonian structures in the hepato duodenal ligament and at the hilum of the liver as observed during hilar dissection.

While removing the connective tissue along the free margin of the hepato duodenal ligament, the first structure to become visible is the common bile duct, with the appropriate hepatic artery on the medial side. These two structures overlie the portal vein. The hepatic artery is the first of these structures to ramify in the ligament, the course of the right branch of the artery usually being underneath the common bile or common hepatic duct, as the case may be, and gives off the cystic artery towards the gall bladder. Mostly two, occasionally three, or even four branches of the right hepatic artery can be dissected at the hilum, more often than not, all of them are situated over the corresponding bile ducts, which, in their turn, run on top of the right portal vein trunk.

The vena portae communis divides upon entering the hilum near the right hilar margin into the two main divisions, the truncus dexter and truncus sinister venae portae.

Extrahepatically the division of the truncus dexter into two large rami can be visualized following retraction of the overhanging right hilar margin or the margins of the incisura dextra as represented in fig 25.

The first ramus usually runs as far as it can be visualized

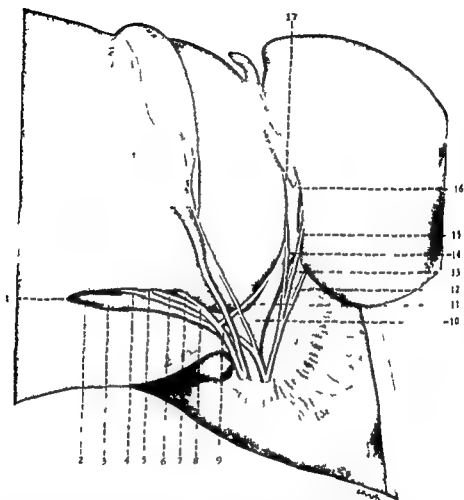


Fig 25 Hilum after removal of the Clissonian capsule (Surgical approach)
 1 vena dextro-caudalis 2 incisura dextra 3 vena dextro-cranialis 4 arteria
 dextra curving around the basis of the vena paramedialis dextra 5 arteria
 paramedialis dextra 6 ductus dexter 7 ductus paramedialis dexter 8 ductus
 hepaticus dexter 9 ductus hepaticus communis 10 truncus sinister vena
 portae 11 ductus hepaticus sinister 12 arteria cranialis I sinistra 13 arteria
 caudalis lobi sinistri 14 arteria paramedialis sinistra 15 vena cranialis I
 sinistra 16 vena caudalis lobi sinistri 17 vena paramedialis sinistra caudalis

in a ventro caudal direction, the second ramus runs in a lateral direction

At the bottom of the sulcus transversus the length of which varies from 2 to 5 cm, there comes the truncus sinister venae portae "pars transversa trunci sinistri venae portae" (Elias & Petty, 1952), which can be dissected

Gradually to the pars transversa we find the left hepatic duct which lies within some millimetres of the hepatic tissue towards the site of its junction with the right hepatic duct however this distance ranges between 0.5 and 5 cm Here, too, retraction of overhanging liver portions facilitates the exposure of the junction The connective tissue at the hilum, which is generally quite loose in consistency, may nevertheless show local condensation between the inferior margin of the caudate lobe and the junction of the hepatic ducts in that event it may hamper their proper dissection

The left hepatic artery is much more superficially located than the left hepatic duct or the truncus sinister venae portae It can be followed without any difficulty up to the point where the different segmental branches penetrate into the liver parenchyma

The arterial branches become visible first of all but further dissection also reveals the portal vein branches running to the caudate lobe Next, a separate arterial branch was often seen running to the quadrate lobe and usually two other branches which disappeared into the left liver lobe

The pars transversa trunci sinistri venae portae curves at an angle of 95–125° in a caudal direction, forming the sagittal part located in the umbilical fossa (recessus umbilicalis of Rex) This part is easily accessible from the dorsal liver surface after removal of the overlying connective tissue

A large portal vein branch arises at the curvature and can be followed together with its corresponding artery occasionally also with the corresponding bile duct which, if it runs ventrally to this vein remains invisible for a distance

of 0.5-1.5 cm after leaving the body of the left liver lobe

The sagittal part of the left portal vein trunk ends abruptly in a bag like structure, indicated as *saccus*

Two groups of vessels can be seen to arise from the *saccus* namely a group originating at the medial aspect, running to the quadrate lobe, and a group arising at the lateral aspect, disappearing into the caudal part of the left liver lobe. All these structures are accompanied by their corresponding arterial branches and bile ducts

Occasionally a bar of liver tissue overlies the *saccus* and has to be divided to give access to this region

It is thus that the different Glissonian pedicles can be visualized at the hilum

Before we compare the relationship between the various Glissonian pedicles which have been visualized at the hilum and the lobes and segments of the intrahepatic Glissonian system, we must consider the common denominator of the different variations in the intrahepatic course of the three structures of the Glissonian system as encountered in our corrosion specimens. In doing so, we shall confine our description to the branches of the first and second order, both from the portal vein, hepatic artery and from the bile ducts

As we have seen, the territory of the left portal vein trunk is sharply marked off from that on the right and does not coincide with the left liver lobe. The left portal vein trunk of which the main ramifications show very few variations in the different casts, consists of two parts, a pars transversa and a pars sagittalis, corresponding with those previously visualized at the hilum, the latter is the remnant of the fused left vitelline and left umbilical vein (fig. 26). From the transverse part, length 2-5 cm, two to three small branches can be seen running cranially towards the caudate lobe, more in particular to the dorsal part of the *lobus paramedialis dorsalis sinister renae caudatae sinistrae* (fig. 27)

Occasionally a rimus can be distinguished at the inferior aspect of the transverse part running into the caudal part of the left paramedian lobe *vena paramedialis sinistra caudalis superior*



Fig 26 Portal vein specimen ventral aspect

1 truncus sinister 2 pars sagittalis 3 vena paramedialis sinistra caudalis
4 vena paramedialis sinistra cranialis 5 vena caudalis lobi sinistri 6 vena cranialis lobi sinistri 7 truncus dexter 8 vena paramedialis dextra 9 vena dextra

The transverse part curves at an angle of 95–125° in a caudo lateral direction forming the sagittal part, located in the dorsal part of the left interlobar fissure. In the foetus the ductus venosus (Arantii) arises from this curvature, directed towards the inferior vena cava.

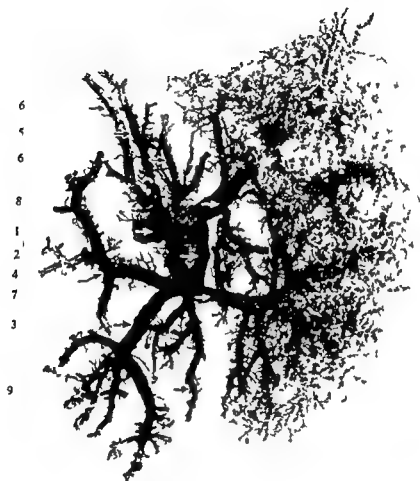


Fig 27 Truncus sinister venae portae with its ramifications ventral aspect
 1 truncus sinister 2 pars sagittalis 3 4 and 5 venae paramediales sinistae
 6 venae caudales 7 vena caudalis lobi sinistri 8 vena cranialis lobi sinistri
 9 vena parumbilicalis

At the site of the caudal bend, a large branch arises at the convex surface of the curvature, running in a latero cranial direction the *vena cranialis lobi sinistri*. Its course is fairly straight. When traversing the cranial part of the left liver lobe, this branch is close to the dorsal surface of the liver and can be reached easily from the dorsal aspect. The ramifications of this vessel correspond with the *segmentum cranio dorsale lobi sinistri*.

The sagittal part ends abruptly in a bag like structure, which was described previously as the saccus. Several vessels arise here: the first, as a group, originating at the medial aspect of the saccus: the *venae paramediales sinistrae caudales*. The ramifications of this group of vessels supply the quadrate lobe almost entirely and coincide with the caudal part of the lobus paramedialis sinister. The second vessel, as a large single branch, though occasionally as several branches, arises at or along the lateral aspect: the *vena intermedialis* and *vena caudalis lobi sinistri*. The ramifications of these vessels correspond with the *segmentum ventro caudale lobi sinistri*.

One branch usually arises from the ventral aspect of the pars sagittalis: sometimes, however, from the medial aspect at a variable point located between the margo inferior of the saccus and the curvature, *vena paramedialis sinistra cranialis*, supplying the cranial and ventral part of the lobus paramedialis sinister.

The *truncus dexter venae portae* shows more variations than the left portal vein trunk. Here we only will describe the course of this vessel as it was usually encountered in our specimens: in a subsequent chapter we shall describe the most prominent variations seen in our casts.

This vessel runs transversely for a variable distance ranging from 0-3 cm before it bifurcates into two rami prior to their disappearance into the body of the right half of the liver (fig. 28).

The first branch the *vena paramedialis dextra*, is of considerable

THE GLISSONIAN SYSTEM



Fig. 8 Truncus dexter venae portae with its ramifications, ventral aspect
 1 vena paramedialis dextra 2 vena dextra 3 vena cranialis lobi dextri 4 vena caudalis lobi dextri 5 vena paracystica
 fan branches 6 vena dextra 7 vena paramedialis dextra

calibre and usually runs in the same direction as the *vena portae communis* Glisson referred to it as (*ramus*) *quintus anterior* (a fifth anterior branch), *Rex* as *ramus ascendens* which as *ramus cysticus* takes a variable origin in the different mammalian livers, a feature also found in the human liver by Deve (1907) to which we shall revert in the next chapter.

This vein, occasionally these veins the arborization of which corresponds with the territory of the *lobus paramedialis dexter* usually runs in a ventro caudal direction in fresh livers.

The ramuli arising from this vessel vary in size, number and direction, a fact which may have an influence upon the size and shape of this lobe as such (figs 12, 13 and 14).

The general picture presented by these variations is two fan shaped groups of ramifications, one at a variable distance (0-3 cm) from its origin, for which we coined the name *dorsal fan* and the second from the distal part of this vein called by us the *ventral fan*. A feature worthy of note is that usually the various veins of the dorsal fan run practically parallel to those of the ventral fan. The latter arise, as it were, at the same spot from the distal end of the right paramedian vein. Only rarely are there branches of the dorsal fan situated posteriorly to such an extent that they prove to arise at or proximal to the origin of the *vena paramedialis dextra* such in contrast to the branches of arteries and bile ducts as will be shown in the next chapter.

The cranial group of veins preponderate over those of the caudal group. The arborization of the ventral fan is sharply marked off from that of the dorsal fan by the *fissura intermedia* (figs 12, 13 and 14).

The right portal vein trunk distally to the point of origin of the right paramedian vein is called *vena dextra*. This branch runs close to the dorsal fan on the liver surface.

Usually one single branch distal to the origin of the *ramus paramedialis dexter* at a distance ranging from a few millimetres to 2 cm or sometimes two distinct separate branches

arises from the inferior aspect of the *vena dextra*, running in a cranial direction to the right of the lateral fossa cystica and *vena paracystica*. The area of ramification of this vein forms that part of the lobus dexter adjacent to the fossa cystica.

The ramus dexter divides into two branches, one to the segmentum dextro craniale, the *vena dextro-cranialis* or *vena cranialis lobii dextri*, and one to the segmentum dextro caudale, the *vena dextro-caudalis* or *vena caudalis lobii dextri*. Several small branches, or one large one, originate from the *vena dextro-caudalis* right at, or slightly distal to, the site of bifurcation of the *vena dextra*, running towards the right liver margin the *vena dextro-narginalis*.

A number of minor branches supply the lobus paramedialis dexter dorsalis.

Inside the liver the portal vein serves as carrier and guides the hepatic arteries and bile ducts. These arteries and bile ducts adhere to these veins just like vines climbing along a pole. Because these structures take a similar intrahepatic course to the portal vein branches supplying or draining the same sectors is distinguished for the ramifications of the portal vein, the arteries and bile ducts are given names identical to the corresponding branches of the portal vein, e.g., *vena paramedialis dextra*, *arteria paramedialis dextra*, *ductus paramedialis dexter* (fig. 29 and 30).

As noted previously, the proper hepatic artery is situated on the medial side of the portal vein. After its bifurcation into a left and right branch, the left hepatic artery runs cranially to the portal vein. Small arteries arise from its trunk running into the caudate lobe. Usually there is one, but occasionally there are several branches to the left paramedian lobe. In the fossa umbilicalis the left hepatic artery divides into a cranial branch to the segmentum cranio dorsale lobii sinistri, and a cranial one to the segmentum ventro caudale lobii sinistri. The right hepatic artery gives off the cystic artery and just before



Fig 11 The three structures of the Glissonian system (intrahepatically)
1 bile duct 2 hepatic artery 3 portal vein



Fig 30 Dorsal aspect of specimen in which bile ducts (white) and left hepatic artery (dark) are injected

entering the body of the liver, or at the right margin of the porta hepatis, it usually bifurcates into two branches, one directed latero cranially, the arteria(lobi)dextra, and the other directed latero caudally, the arteria paramedialis dextra

The pattern of division of the *bile ducts* is generally fairly similar to that of the arteries. The left hepatic duct is composed of a ductus cranialis lobi sinistri, originating in the dorso cranial left lobar segment, which joins the ductus caudalis lobi sinistri ventral to the curvature of the left portal vein, receiving one duct from the left paramedian lobe and 2-3 ductules from the caudate lobe. The right hepatic duct is usually composed of two bile ducts. The first drains the right lobe and is formed by the confluence of the dextro cranial and dextro caudal ducts, which together form the right lobar duct. The second drains the right paramedian lobe. They unite at the hilum, usually outside the body of the right liver at the right margin of the porta hepatis. After its junction the right hepatic duct receives a variable number of minor branches from the caudate lobe and then joins the left hepatic duct, thus forming the main hepatic duct. After its junction with the cystic duct it is called the common bile duct or ductus choledochus, which is located laterally to the portal vein.

In concluding this chapter we can say on the basis of a comparative study of the hilar dissection with the injection corrosion specimens, that the two Glissonian pedicles, which can be identified in the former in the right hilar area, are identical with the right paramedian lobar and right lobar structures.

Moreover the *Glissonian pedicles* which can be dissected in the left hilar area, as well as the umbilical fossa, are identical with the different segmental structures of the segments previously described.

Every lobar or segmental pedicle can be dissected in the left hilar area and the umbilical fossa except that for the ventro cranial part of the left paramedian lobe.

Chapter IV

VARIATIONS IN THE STRUCTURES OF THE GLISSONIAN SYSTEM

The first part of this chapter will be concerned with the variations which occur in the structures of the Glissonian system outside the liver, and more especially with those found at the hilum of the liver. Wherever possible, our findings will be collated with the data published in the literature, but this has not always been feasible on account of the fact that in some respects we have found it necessary to depart from the conventional approach to the matter, mainly because, in the preceding section, we adopted a certain classification and nomenclature not wholly identical with those used by other investigators, notably Healey *et al* and Couinaud. This is not to say that we are less aware than they of the importance of these variations to surgery, and that they are undoubtedly factors to be reckoned with in surgical intervention.

The variations of the different structures will be dealt with systematically for each individual structure, our aim being to define certain patterns into which the various structures fall at the hilum of the liver in relation to the lobes and segments drained or supplied by them.

Blood is discharged from the stomach, pancreas, intestines and spleen into the portal vein, which is composed of the following vessels:

The *vena coronaria sinistra*, which runs alongside the lesser curvature of the stomach near the *arteria gastrica sinistra*. As

demonstrated by Douglass *et al* and Gilfillan in their dissections (both in 1950), this vessel may discharge into the vena lienalis (58.9%) the vena portae communis (24.4%) or the vena mesenterica superior (16.7%). In the first mentioned case this vessel enters the vena lienalis at the site of the latter's discharge into the vena portae. (All these and subsequent figures are cited from Douglass *et al*, 1950)

The vena mesenterica magna *sive superior*, which drains the blood from the region served by the arteria mesenterica superior into the vena portae. This vein is composed of the vena pancreatico-duodenalis the vena gastro-epiploica dextra, the vena ileo colica, the vena colica dextra and the vena colica media. Here too variations are liable to occur. Thus Douglass *et al* found a vena pancreatico duodenalis in their series which consisted of a single vessel discharging into the vena portae communis (38%) or of several vessels with one or more discharging into the vena portae and the others into the vena mesenterica superior. They sometimes saw the vena gastro-epiploica discharging into the vena portae (2.2%) or else into the vena lienalis (2.2%).

The vena lienalis which drains the blood from the stomach via the coronary vein in only 16.7% of dissections. They also found that in 38% of the cases the vena mesenterica inferior discharged into this vessel.

The vena mesenterica parva *sive inferior* which drains the blood from the region supplied by the arteria mesenterica inferior and which is connected with the vena hypogastrica via the vena haemorrhoidalis superior via the plexus haemorrhoidalis. The vena mesenterica inferior usually enters the medial one third of the splenic vein but may enter the superior mesenteric vein. In 38% of their dissections Douglass *et al* saw this vessel discharging into the vena lienalis in 29.3% of the cases into the vena mesenterica superior and in 32.7% together with the vena lienalis and the vena mesenterica superior, into the vena portae communis.

On the evidence of the above figures it is clear that the site of entry of the many branches of which the vena portae is composed varies considerably.

Glenard (1890) believed that blood is supplied to various areas of the liver from certain sections of the spleen, gall bladder, pancreas and gastrointestinal tract. In his view the left half of the liver receives blood from the spleen, stomach, duodenum, colon descendens, sigmoid and pancreas, the right half of the liver being supplied from the remainder of large and small guts. Serége (1901), Copher and Dick (1928) and Mermans (1936) explained this feature on the basis of the different separate blood currents present side by side in the portal vein. The localization of pathology in the liver of an infectious or malignant nature transmitted to the liver from those organs which drain their blood via the portal vein has been linked with this stream-lined effect of the portal vein blood. There are examples in pathology which illustrate the fact that predictions as to where a secondary lesion will be manifested in the liver are exceedingly precarious. Hence seen in this light the above variations acquire a clinical significance as well and show yet again why it is often impossible to make any such prediction (see Chapter VII).

The venous collateral circulation of the liver is far less extensive than what is generally regarded as the arterial collateral circulation, as will presently appear. As far as the collateral circulation is concerned, we found anastomoses between the intrahepatic branches of the portal vein and the subserosal tiny vessels of the gall bladder. It was also possible to demonstrate anastomoses with the venous channels of the diaphragm by way of the triangular and coronary ligaments.

As a rule, there is little variation to be seen in the distribution of the vena portae at the hilum of the liver. The most important exception has already been noted by Rex, viz. that of the origin of the vena paramedialis dextra. In the majority of mammalian livers this vessel was found to arise from the truncus sinister venae portae, usually from the right trunk in human livers, but sometimes this vessel arose exactly at the bifurcation of the vena portae communis. Of 30 specimens dissected by Deve (1907) 22 showed this vessel deriving from the right trunk, 3 from the left and 5 from the bifurcation.

We found the following percentages in our specimens. In

70% this vessel proved to derive from the right trunk, in 10% from the left and in 20% from the bifurcation

Let us now consider other variations which are liable to occur at the hilum

In the case of the *fossa umbilicalis* it has been found that it is not possible to dissect every individual branch of the portal vein here. The branches which arise on the medial and lateral side of the sacculus, are not invariably the only vessels supplying the areas on either side of the sacculus. Some of them, varying in number also arise at the ventral ventro lateral and ventro-medial surface of this structure

As we hope to show in the second part of this chapter the vessels supplying the cranial section of the left lobus paramedialis* can seldom be dissected in the fossa as these likewise arise in the ventral aspect of the pars sagittalis venae portae sinistrae

We have also though more rarely come across a curious variation in that of the vena cranialis lobii sinistri arising at the same level as the vena caudalis lobii sinistri in fact we once found the former branch deriving from the latter though still within the region of the fossa umbilicalis!

As to the right hilum it may be said that in almost all livers in which the vena paramedialis dextra derives from the truncus dexter venae portae this vessel can be visualized either in the hilum or in the incisura dextra after dissection

The procedure to be adopted in the event of the absence of the incisura dextra will be discussed in Chapter VIII

The vena paramedialis dextra usually runs caudo ventrally in fresh livers. Often the vena paracystica which commonly arises somewhat more distally cannot be visualized in this way

Not only do variations occur at the site of origin of the hepatic artery but also in the number of arterial branches sup-

* When we speak of the lobus paramedialis we mean the ventral lobes our references to the Glissonian territory of the caudate lobe are qualified by the adjective dorsalis

plying the liver as well as the site of division of the proper hepatic artery

Von Haller (1764) and Hyrtl (1873) pointed out that injection of the aorta, following ligation of the proper hepatic artery in the hepato duodenal ligament, resulted in a filling of a great number of minor arterial branches running to the liver. These branches were called "*arteriae hepaticae accessoriae*". Initially all these vessels were considered as arterial "*collaterals*". They were seen to arise from the aorta, superior mesenteric artery, left gastric artery, right renal or spermatic artery, right phrenic artery, internal mammary artery, pericardiophrenic artery, inferior epigastric artery, etc (Henle, 1864).

Von Haller even thought that the *arteria hepatica accessoria ex mesenterica* was always present "*perpetua arteria est, quae ex mesenterico trunco omnium prima (ad hepate) prodit*", ("there is always an artery present which arises from the mesenteric artery and goes to the liver"), which was partly refuted by Hyrtl's observation in 20 injected cadavers of infants, where he found this artery to be present thirteen times. The occurrence of the other accessory arteries was even less frequently observed.

Hyrtl also observed that occasionally the accessory hepatic artery may develop to such an extent '*dass sie normale Aeste der Leberarterie uberflussig machen und ganzlich verdrangen*' ("that they make the normal branches of the hepatic artery superfluous and completely displace them"). This is readily comprehensible from the ontogenetic standpoint. Hence this vessel is an "*aberrant hepatic artery*".

In over 250 cadavers Rio Branco (1912) found the existence of one single hepatic artery in 59%, arising in 55% of cases from the celiac artery and in 4% from the superior mesenteric artery. Occasionally the right and left rami arise separately from the various upper abdominal arteries, such as superior mesenteric artery or left gastric artery. Rio Branco traced the site of origin in the case of a double hepatic artery, which

occurred in 40% of his dissections, and found a right branch arising from the superior mesenteric artery in 10%, a left branch arising from the left gastric artery in 10%, a reduplication of the proper hepatic artery in 20% of his dissections

Henle (1864) described cases with *three* separate hepatic arteries, one from the left gastric artery, the second from the coeliac artery, and the third from the superior mesenteric artery. According to this author this variation would occur in a small percentage of all dissections, which was confirmed by Rio Branco who found three hepatic arteries in 2% of his dissections taking the same origin as described by Henle

The presence of these so called accessory hepatic arteries has also been described by Haberer (1905). He noted that their number, site of origin and course are variable. These vessels occurred in 32% of 75 dissection specimens which were at his disposal, sites of origin mentioned are those previously mentioned by Von Haller: aorta, superior mesenteric artery, left gastric artery, right renal or spermatic artery, etc. etc.

In our casts we were able to confirm the observation made by Hyrtl (1873) and Healey *et al* (1953) that these accessory or replacing arteries *do not supply areas already receiving blood from the proper or the aberrant hepatic artery*. These accessory hepatic arteries which enter the liver at the porta hepatis, umbilical fossa or occasionally at the dorsal surface of the organ without entering the hilum namely by way of adhesions between the liver and the hepato-duodenal ligament, the coronary and hepato renal ligaments are therefore endarteries in the sense originally described by Cohnheim (1876) and do not serve as collaterals in cases of occlusion of the proper hepatic artery (Hyrtl, 1873, Haberer, 1905, Healey *et al* 1953).

As regards the hepatic arteries anastomoses were occasionally observed in the umbilical fossa between the arteria paramedialis sinistra and the arteria caudalis lobi sinistra usually ventrally to the saccus or the pars sagittalis venae portae.

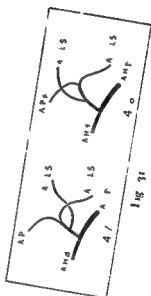
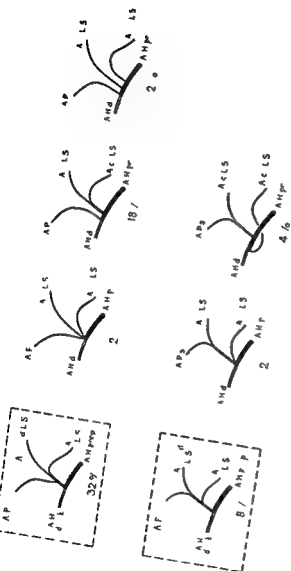
Segall (1923) described numerous anastomoses between the

intrahepatic branches of the hepatic artery and the phrenic artery. These shunts, first observed by Cruveilhier (1867), which pass via the ligamentum triangulare et coronarium to the liver and anastomose with the arterial vessels in the capsule of the liver, are vessels of the 6th–7th order of subdivision. In contrast to the accessory artery, it has been reported that, after occlusion of the proper hepatic artery, these vessels, serving as collateral channels, can maintain a compensatory arterial circulation (Haberer, 1905) after an initial period of a week to ten days (Segall, 1923, Markowitz, 1949).

In the hepato duodenal ligament, the proper hepatic artery is situated on the medial side of the common duct, overlying the portal vein. Dissection specimens show a variable point of division into right and left hepatic artery. In most of the specimens the bifurcation is in the porta hepatis near the border of the quadrate lobe. Segall (1923), who investigated the location of the bifurcation of the proper into right and left hepatic artery, found the point of division at the former site to be present in 40% of his 55 dissections. In Healey's as well as our specimens the bifurcation in this area is always situated to the left of the main fissure. In about 35% of Segall's specimens the bifurcation was present between porta hepatis and inferior margin of the quadrate lobe, and in about 25% at a variable point between inferior liver margin and coeliac axis.

Both right and left hepatic artery usually subdivide into two or more branches at the hilum. Descomps and Flourens (1910) gave the following percentages for the different numbers of branches, which were visualized at the hilum in 50 dissections:

right hepatic artery		left hepatic artery	
1 branch	4%	1 branch	22%
2 branches	26%	2 branches	36%
3	42%		
4	16%	4	12%
5	6%	5	8%



The *arteria hepatica sinistra* may divide in a number of ways. Among the fifty corrosion specimens in which we studied the variations presented, we found nine patterns where the left hepatic artery divided in the hilum (fig 31)

The pattern usually encountered in our specimens is consistent with the situation as represented in fig 32. The left hepatic artery is directed medially, overlying the pars transversalis venae portae sinistrae, and is the first structure encountered during the left hilar dissection. It gives off a branch (diameter of the lumen 2-3 mm) for the left paramedian lobe, i.e., the left paramedian artery, which can be followed extrahepatically to the umbilical fossa as it runs along the medial surface of the sagittal part of the left portal vein trunk. Between the origin and the site of entry into the liver of this vessel, it throws out a branch, usually at the level of the curvature of the left portal vein trunk, this, running in a central cranial direction, almost immediately disappears into the body of liver tissue, joining the left paramedian cranial vein (see pg 88). It supplies the cranial part of the left paramedian lobe with arterial blood. The sagittal arterial branch, running along the medial surface of the pars sagittalis venae portae sinistrae, joins the corresponding vein arising from the medial aspect of the saccus before entering the body of liver tissue, after which these structures continue their intrahepatic course in close proximity. Distally to the origin of the left paramedian artery, the left hepatic artery, usually in the umbilical fossa, occasionally medial to or otherwise dorsal to the portal vein, divides into two branches, which pass either over or laterally to the pars sagittalis. These two branches, the cranial and caudal left lobar arteries, join the corresponding veins arising from the lateral aspect, at the curvature and at the saccus of the pars sagittalis before entering the left liver lobe, this can be visualized by incising the capsule along the left liver lobe (see fig 25)



Fig 32

The venae cranialis et caudales lobii sinistri come into view at the site where these vessels arise from the pars caudalis venae portae, and are accompanied by the arterial branches.

A modification of this condition is shown in fig. 33, where no distinct left hepatic artery appears to be present. Instead of this vessel, three arterial branches arise at the same level from the proper hepatic artery, which together supply the hepar sinister. These branches are the arteria paramedialis ventralis sinister, the arteria cranialis and the arteria caudalis lobii sinistri (2%).



Fig. 33

Another recurrent variation of the normal pattern, which was encountered in 18% of the corrosion specimens (Healey *et al.* 25%), is represented by fig. 34. The left hepatic artery alone supplies the lobus sinister, this vessel is broken up into

an arteria cranialis and an arteria caudalis lobii sinistri. The arteria paramedialis sinistra originates distally to the site of origin of the left hepatic artery, or left lobar artery, as the case may be, and therefore ought to be regarded as arising from the right hepatic artery.



Fig. 34

A modification of this condition appears in fig. 35. Apparently, there is no distinct left hepatic artery.



Fig. 35

All three branches arise separately from the proper hepatic artery (2%).

The other extreme as represented by fig. 36 shows that the left paramedian artery is a branch of the most distal part of the left hepatic artery which bifurcates at the level of the saccus into an arteria

caudalis lobii sinistri and an arteria paramedialis sinistra. The arteria cranialis lobii sinistri originates at a more proximal point from the left hepatic artery (9%) (Healey *et al.* 35%).

The *arteria hepatica sinistra* may divide in a number of ways. Among the fifty corrosion specimens in which we studied the variations presented, we found nine patterns where the left hepatic artery divided in the hilum (fig. 31).

The pattern usually encountered in our specimens is consistent with the situation as represented in fig. 32. The left hepatic artery is directed medially, overlying the pars transversalis vene portae sinistae, and is the first structure encountered during the left hilar dissection. It gives off a branch (diameter of the lumen 2-3 mm) for the left paramedian lobe, i.e., the left paramedian artery, which can be followed extrahepatically to the umbilical fossa as it runs along the medial surface of the sagittal part of the left portal vein trunk. Between the origin and the site of entry into the liver of this vessel, it throws out a branch, usually at the level of the curvature of the left portal vein trunk, this running in a central cranial direction, almost immediately disappears into the body of liver tissue, joining the left paramedian cranial vein (see pg. 88). It supplies the cranial part of the left paramedian lobe with arterial blood. The sagittal arterial branch, running along the medial surface of the pars sagittalis vene portae sinistae, joins the corresponding vein arising from the medial aspect of the sacculus before entering the body of liver tissue, after which these structures continue their intrahepatic course in close proximity. Distally to the origin of the left paramedian artery, the left hepatic artery, usually in the umbilical fossa, occasionally medial to or otherwise dorsal to the portal vein, divides into two branches which pass either over or laterally to the pars sagittalis. These two branches, the cranial and caudal left lobar arteries, join the corresponding veins arising from the lateral aspect, at the curvature and at the sacculus of the pars sagittalis before entering the left liver lobe. This can be visualized by incising the capsule along the left liver lobe (see fig. 25).



Fig. 32

The *venae craniales et caudales lobi sinistri* come into view at the site where these vessels arise from the *pars sagittalis venae portae*, and are accompanied by the arterial branches.

A modification of this condition is shown in fig 33 where no distinct left hepatic artery appears to be present. Instead of this vessel, three arterial branches arise at the same level from the proper hepatic artery, which together supply the *hepar sinister*. These branches are the *arteria paramedialis ventralis sinister*, the *arteria cranialis* and the *arteria caudalis lobi sinistri* (2%)



Fig 33

Another recurrent variation of the normal pattern, which was encountered in 18% of the corrosion specimens (Healey *et al* 25%), is represented by fig 34. The left hepatic artery alone supplies the lobus sinister; this vessel is broken up into

an *arteria cranialis* and an *arteria caudalis lobi sinistri*. The *arteria paramedialis sinistra* originates distally to the site of origin of the left hepatic artery or left lobar artery, as the case may be, and therefore ought to be regarded as arising from the right hepatic artery.



Fig 34

A modification of this condition appears in fig 35. Apparently there is no distinct left hepatic artery. All three branches arise separately from the proper hepatic artery (2%).



Fig 35

The other extreme as represented by fig 36 shows that the left paramedian artery is a branch of the most distal part of the left hepatic artery which bifurcates at the level of the saccus into an *arteria caudalis lobi sinistri* and an *arteria paramedialis sinistra*.

The *arteria cranialis lobi sinistri* originates at a more proximal point from the left hepatic artery (8%) (Healey *et al* 35%).

The *arteria hepatica sinistra* may divide in a number of ways. Among the fifty corrosion specimens in which we studied the variations presented, we found nine patterns where the left hepatic artery divided in the hilum (fig. 31).

The pattern usually encountered in our specimens is consistent with the situation as represented in fig. 32. The left hepatic artery is directed medially, overlying the *pars transversalis venae portae sinistrae*, and is the first structure encountered during the left hilar dissection. It gives off a branch (diameter of the lumen 2-3 mm.) for the left paramedian lobe, i.e., the left paramedian artery which can be followed extrahepatically to the umbilical fossa as it runs along the medial surface of the sagittal part of the left portal vein trunk. Between the origin and the site of entry into the liver of this vessel it throws out a branch, usually at the level of the curvature of the left portal vein trunk, this running in a central cranial direction, almost immediately disappears into the body of liver tissue, joining the left paramedian cranial vein (see pg. 88). It supplies the cranial part of the left paramedian lobe with arterial blood. The sagittal arterial branch running along the medial surface of the *pars sagittalis venae portae sinistrae* joins the corresponding vein arising from the medial aspect of the saccus before entering the body of liver tissue after which these structures continue their intrahepatic course in close proximity. Distally to the origin of the left paramedian artery, the left hepatic artery usually in the umbilical fossa, occasionally medial to or otherwise dorsal to the portal vein divides into two branches which pass either over or laterally to the *pars sagittalis*. These two branches the cranial and caudal left lobar arteries join the corresponding veins arising from the lateral aspect, at the curvature and at the saccus of the *pars sagittalis* before entering the left liver lobe. This can be visualized by incising the capsule along the left liver lobe (see fig. 25).



FIG. 3

The *venae craniales et caudales lobii sinistri* come into view at the site where these vessels arise from the *pars sagittalis venae portae*, and are accompanied by the arterial branches.

A modification of this condition is shown in fig 33 where no distinct left hepatic artery appears to be present. Instead of this vessel, three arterial branches arise at the same level from the proper hepatic artery, which together supply the *hepar sinister*. These branches are the *arteria paramedialis ventralis sinister*, the *arteria cranialis* and the *arteria caudalis lobii sinistri* (2%)

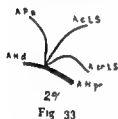


Fig 33

Another recurrent variation of the normal pattern, which was encountered in 18% of the corrosion specimens (Healey *et al* 25%) is represented by fig 34. The left hepatic artery alone supplies the lobus sinister, this vessel is broken up into

an *arteria cranialis* and an *arteria caudalis lobii sinistri*. The *arteria paramedialis sinistra* originates distally to the site of origin of the left hepatic artery, or left lobar artery, as the case may be, and therefore ought to be regarded as arising from the right hepatic artery.



Fig 34

A modification of this condition appears in fig 35. Apparently, there is no distinct left hepatic artery.

All three branches arise separately from the proper hepatic artery (2%).

The other extreme as represented by fig 36 shows that the left paramedian artery is a branch of the most distal part of the left hepatic artery which bifurcates at the level of the saccus into an *arteria caudalis lobii sinistri* and an *arteria paramedialis sinistra*.

The *arteria cranialis lobii sinistri* originates at a more proximal point from the left hepatic artery (8%) (Healey *et al* 35%)

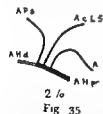
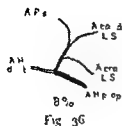


Fig 35

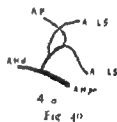


From the modification represented in fig 37 it appears that the latter may even arise from the proper hepatic artery (2%) A modification of this condition is represented by fig 38 Here the arteria hepatica sinistra supplies the left paramedian lobe and the ventro crural segment of

the left liver lobe The cranial segment artery arises from the right hepatic artery (4%)

Two rather unusual variations are reproduced in figs 39 and 40, the arteria crudis lobi sinistri arises proximal to the arteria cranialis (4%) (see fig 39) and in fig 40 the arteria paramedialis sinistra arises from the arteria cranialis lobi sinistri (4%)

Together these variations represent 76% of our corrosion specimens In the other 24%, regions normally supplied by



the left hepatic artery did not fill probably on account of the fact that these areas were supplied by accessory hepatic arteries

In 14% of the specimens the arterial bed of the hepatis sinister did not fill up in 8% the arteries of the left lobe were not filled in 2% the arteries of the left paramedian lobe remained empty (fig 41)

Several patterns of ramifications of the right hepatic artery can

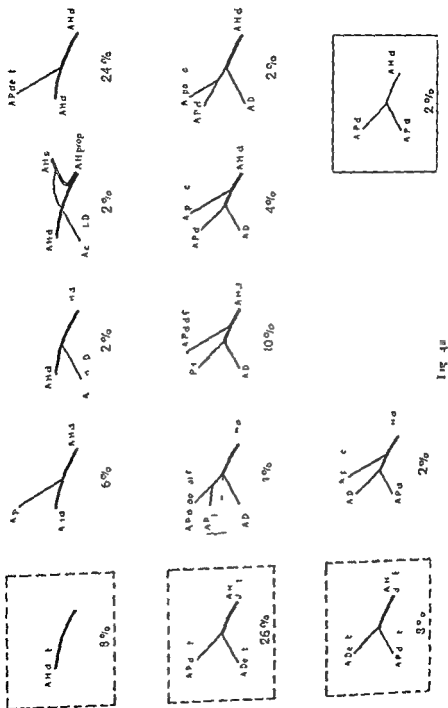
as a rule be observed at the hilum. In 86% of the specimens the arterial branches are located dorsally to the bile ducts (Herley 87%) and appear as the first structures during hilar dissection. In 8% of the specimens (Herley 11%) they passed in front of the ducts. In 6% of the specimens it was impossible to tell definitely whether they were situated dorsally or ventrally relative to the bile ducts. Distally to the division of the right hepatic artery the different arterial branches join the corresponding veins. In at least 22% of the specimens the right hepatic artery divided into more than two branches at the hilum; however we could not ascertain definitely in our



crysts whether a further division took place at the hilum or intrahepatically. For this reason we assume that the further divisions take place intrahepatically.

We distinguish three basic patterns for the division of the right hepatic artery: 1. the first characterized by an artery which without dividing penetrates into the right half of the liver; the second characterized by the division of the right hepatic artery into a right paramedian artery directed caudo laterally and a right artery directed cranio laterally; finally the third which represents the reverse of the second group in that the caudal branch is the *arteria dextra* instead of the *arteria paramedialis dextra* while the cranial branch runs towards the right paramedian lobe¹ (fig. 42).

The first pattern is characterized by the fact that the right hepatic artery enters the liver undivided (8%) (fig. 43).



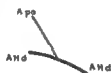
Frequently a minor branch arises from the right hepatic artery, but it appears to be a segmental instead of a lobar or terial trunk.

This pattern was encountered as such in 3½% of the specimens. In 10% of the specimens the minor branch appeared to supply one of the segments of the lobus dexter viz, in 6% the cranial segment (fig 43) or a part of this segment (or else ramifications of the vena portocystica) and in 2% the cranial segment (fig 45). In 2% of the specimens the right hepatic artery without dividing at the hilum supplied the whole



8%

Fig 43



6%

Fig 44



2%

Fig 45

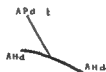
right half of the liver minus the cranial segment of the right lobe which received arterial blood from the left hepatic artery (fig 46).

In 24% of the specimens (fig 47) a minor branch was seen arising from the right hepatic artery supplying different areas of the right paramedian lobe, in the majority of specimens this branch had a considerable calibre and supplied the right paramedian lobe almost completely.



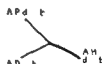
2 /

Fig 46



24%

Fig 47



26%

Fig 48

All the variations of the second group have one feature in common namely that the right hepatic artery divides right at the

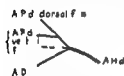
hilum into an artery directed caudo laterally, running to the right paramedian lobe, and another artery, directed cranio-laterally, running into the lobus dexter

The total number of specimens in which we met this condition was 46% (Healey 52%) This condition, without any further variations, was encountered in 26% of the specimens (fig 48)

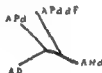
In 4%, a trifurcation instead of a bifurcation of the right hepatic artery was found (fig 49) The branch directed most cranially appeared to be the arterial branch supplying the 'dorsal fan' area, the middle branch supplied the 'ventral fan' area of the lobus paramedialis dexter, the cranial branch supplied the lobus dexter

In 10% of the specimens (fig 50) an arterial branch supplying the 'dorsal fan' area of the lobus paramedialis dexter arose even proximal to the site of the bifurcation of the right hepatic artery

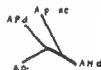
In 4% of the specimens (fig 51) a branch supplying part of



4%
Fig 49

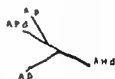


10%
Fig 50



4%
Fig 51

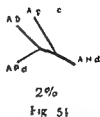
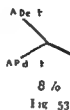
the cranial segment of the lobus dexter, namely the area paramedialis was seen to arise proximally to the bifurcation of the right hepatic artery in 2% of the specimens (fig 52) from the arterial paramedialis dextra



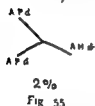
2%
Fig 52

A third group of variations though a minor one considering the number of cases encountered, showed the reverse pattern of the second group the cranio lateral arterial branch, distal

to the bifurcation of the right hepatic artery, appeared to be the arteria dextra, instead of the arteria propria medialis dextra (10%) (fig 53 and 54). In 2% of the specimens a small artery was seen to arise proximal to the bifurcation of the right hepatic artery supplying the area of ramification of the ventral paracystic (fig 54).



In only one specimen did we find the lobus dexter supplied by an aberrant hepatic artery! (fig 55)



While studying the literature, both Hyrtl and Rex were struck by the great number of variations occurring in the ramifications of bile ducts in mammalian livers, which at that

time accounted for a confusion in this field throwing Hyrtl into such despair that he wrote: 'Man verliert allen Bucher glauben und alles Vertrauen auf den Werth von Citaten wenn man auf solche Unrichtigkeiten stösst'. Rex noted the cause of this confusion: 'Diese Verwirrung beruht lediglich in dem einem Umstande dass soviel ich ersehe, meist nur ein Thier dem Untersucher vorgelegen, dass ferner aus dem Grunde dass man sich mit einem gewonnenen Bilde begnugte auch die Erkenntniss der mannigfachen Variabilität der Verastlung der Gallenwege versagt bleibt'. (This confusion rests entirely on the fact that as far as I can see most of the research has been done on one animal, and further on the fact that the research workers were content with the conception they had formed of it. This has made it impossible to acquire a knowledge of the great variability of bile duct ramifications) To a certain extent the same applies to the division of the bile ducts of the human liver.

Rex inferred the existence of three hepatic ducts from his

corrosion specimens, 112, 1 right, left and middle duct

Among those who have studied the topography of the bile ducts were E. Ruge (1909) in 43 and Johnson and Anson (1952) in 35 livers from which the hilum was dissected. Ruge found 1 ductus hepaticus communis in 32 specimens arising from 1 right and left hepatic duct. In 5 specimens he found the ductus hepaticus communis deriving from three, and three times from five hepatic ducts. He also reported the absence of any ductus hepaticus communis at all in three specimens owing to the fact that the cystic duct discharged into the right hepatic duct. Ruge further noted that, where the ductus hepaticus medius was present, it often issued from the quadrate lobe (namely, in five of the nine cases!)

In 35 livers Johnson and Anson (1952) found that one to two ducts 'joined the point of confluence of the main left and right ducts' in five specimens. These tributaries proved to be of far thinner calibre than the "main ducts". A further comment was that the bile ducts are the most ventrally situated structures as a rule in the hilum, and are by no means easy to dissect.

Extrahepatic bile ducts have been described at other sites besides the hilum, 112, the so called aberrant ducts (Ferran 1753, Kiernan, 1833). They are most frequently found in the connective tissue round the inferior vena cava, the coronary ligament, the left cranial margin of the liver, in the umbilical fossa and the hepatogastric ligament. It is possible that ducts of Iuschka (Raport & Hromada, 1950) are such 'aberrant' hepatic ducts. They communicate with the intrahepatic duct system and may be opened during upper abdominal surgery.

Raport *et al* reported two instances in which vagotomy was complicated by cholerrhagia due to opening of one of these ducts. They are absent in the foetus and child, their number increasing with advancing age. Raport *et al* regard these ducts as intrahepatic ducts which have been preserved following reduction or atrophy of liver tissue and have thus become extrahepatic.

The variations at the site of discharge of the hepatic ducts coming from the *left half* of the liver were classified in six groups for the fifty corrosion specimens which we studied. It must be emphasized yet again that we are now dealing with findings in the corrosion specimen as dissection of the bile ducts at the hilum is a major problem! (fig 56)

The most recurrent feature is represented in fig 57. Two ducts can be recognized leaving the lobus sinister at the same

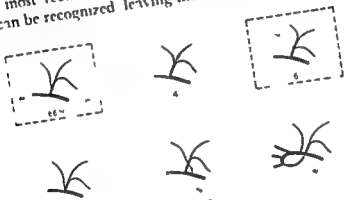


Fig 56

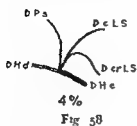
sites where both segmental veins and arteries enter this lobe. The bile ducts which are the most ventrally located structures at the hilum run anteriorly, i.e. ventrally to the vein and artery. In the fossa umbilicalis the ductus caudalis lobi sinistri runs laterally to the pars sagittalis venae portae and joins the ductus cranialis lobi sinistri ventrally to the bend of the left portal vein trunk. The common duct of the left lobe joins the paramedian duct in the hilum. The ductus paramedian sinister leaves the lobe at the site where vein and artery enter their territory at the medial side of the saccus. During its course along the medio ventral surface of the pars sagittalis venae portae a branch from the cranial territory of the lobe joins this duct.



66%

Fig 57

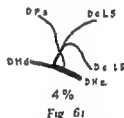
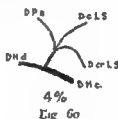
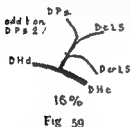
We found this condition in 66% of our specimens, Healey *et al* in 67% of his crabs. Occasionally, bile ducts were seen to cross over between the segmentum crudalis lobi sinistri and the crudal area of the left paramedian lobe.



A union of all three ducts at one site was found in 4% of our specimens (fig 58).

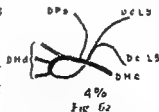
A union of the ductus paramedianus sinister and ductus crudalis lobi sinistri takes place at the level of the sacculus in 16% of our specimens (Healey *et al* 25%) (fig 59).

A union of the ductus lobi sinistri and ductus paramedianus sinister at the site of junction with the right hepatic duct occurs in 4% of the specimens (fig 60).



The presence of the left hepatic duct was not definitely established in 8% of our specimens.

It appears that the ductus lobi sinistri enters the common hepatic duct proximal to the ductus paramedianus sinister in 4% (fig 61). In 4% of the specimens the different bile ducts from the right liver half enter the ductus lobi sinistri before joining the left paramedian duct (fig 62).



The variations at the site of discharge of the hepatic ducts coming from the right half of the liver were arranged in three groups, each of which was subdivided.

In the first of these three groups the right hepatic duct is composed of a cranial right duct and a right paramedial duct, in the second group the arrangement is precisely reversed and in the third the junction of the two lobar ducts has already taken place intrahepatically (fig 63)

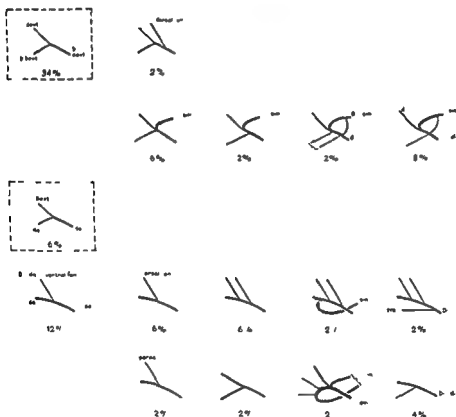
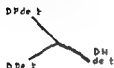


Fig 63

In 54% of the specimens the right hepatic duct is composed of the two lobar ducts u_c that cranially located from the lobus dexter and the caudal duct from the lobus paramedialis dexter. The condition without any further variations was encountered in 34% of the specimens (fig 64). In 6% of the

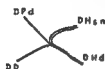
specimens these two ducts unite with the left hepatic duct forming the common hepatic duct (fig 65)

In 2% of the specimens the ductus dexter enters the common hepatic duct, hence there is no right hepatic duct. The ductus paramedialis dexter joins the left hepatic duct (fig 66)



34%

Fig 64



6%

Fig 65

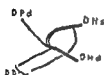


2%

Fig 66

Moreover, in one specimen the bile of the cranial segment of the lobus dexter is discharged into the left hepatic duct (fig 67)

In 8% of the specimens, likewise without the right hepatic duct, the ductus paramedialis dexter joins the left hepatic duct to form the common hepatic duct, while the ductus dexter enters the left hepatic duct (fig 68). In one instance a duct



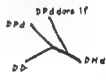
2%

Fig 67



8%

Fig 68



2%

Fig 69

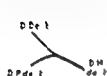
from the dorsal funiculus joined the right hepatic duct distally to the point of junction of both lobar ducts (fig 69)

A second pattern, in which the ductus dexter is located cranially to the ductus paramedialis dexter is only seen in 6% of the specimens (fig 70)

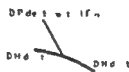
A third pattern, encountered in 38% of our specimens is that in which it proves difficult to visualize both lobar ducts at the hilum; its junction therefore is considered to be intrahepatically. The right hepatic duct leaves the liver at the

right hilar margin. Segmental or territorial branches may join the right hepatic duct extrahepatically.

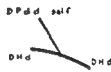
In 12% of our specimens a duct can be recognized joining



6%
Fig 70



12%
Fig 71



6%
Fig 72

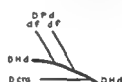
the right hepatic duct which drains the ventral fan area (fig 71), in 6% a distinct separate duct drains the dorsal fan area (fig 72). In 6% of the specimens two ducts can be visualized



6%
Fig 73



2%
Fig 74

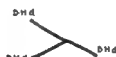


2%
Fig 75

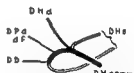
entering the right hepatic duct at the hilum the proximal one from the ventral fan area of the right paramedian lobe the distal one from the dorsal fan area (fig 73).



2%
Fig 76



2%
Fig 77



2%
Fig 78

In 2% of the casts these two ducts join the common hepatic instead of the right hepatic duct (fig 74).

In another 2% two ducts from the dorsal fan area and one

duct from the segmentum craniale lobi dextri join the right hepatic duct (fig 75)

In 2% one duct drains the area paramedialis (fig 76), while in another specimen two ducts on the right join extrahepatically, both draining areas from both lobes (fig 77) Moreover in 2% a ductus dexter and a duct from the dorsal fan area separately enter the left hepatic duct (fig 78), and in 4% the ductus dexter joins the right hepatic duct (fig 79)

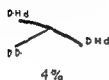


Fig 79

Up to the present, we have been concerned solely with variations that are to be observed at the *hilum* of the liver and it will have become clear that the extent to which the ramifications of the various structures of the Glissonian system can be followed in the left *hilum* is far greater than in the right *hilum*. Accordingly, we had no difficulty in discovering how all the lobes and segments are arterially supplied in the *left* half of the liver and how these areas drain their bile, with one exception, *i.e.*, the ventro cranial region of the left paramedian lobe. The various structures of the Glissonian system at the *hilum* cannot be rendered visible in this part of the liver as they can in the rest of the left liver.

We have already ascertained in the *right* half of the liver that the vena paramedialis dextra and the vena dextra can be dissected at the *hilum* or its extension the incisura dextra. As a rule the corresponding artery and bile duct run along the medial side of the right paramedian vein, but this vessel may be accompanied by several arteries and bile ducts, supplying or draining different areas of the right paramedian lobe. It is then found that these arteries and bile ducts are usually placed laterally and/or cranially and/or caudally relative to the vein and either derive from or run to the right artery or right duct respectively.¹

Something similar occurs in a number of livers as regards the

distribution of the arteries and bile ducts supplying or draining the two segments of the right lobe. Although the natural inference is that the two segments of the right hepatic lobe would be supplied with arterial blood from the right artery and would drain bile via the right duct into the right hepatic duct, apparently this is not an invariable rule.

For this reason we shall touch briefly on these variations in a general way, not dwelling on individual variations, as they do not readily lend themselves to systematic discussion and any such attempt would only lead to confusion.

Lobus paramedialis sinister Cranial area

As has been stated the ventral portal branches for the ventro cranial area of the left paramedian lobe cannot be visualized at the hilum. This area is usually supplied by several branches (see fig. 27, p. 56) originating from the sagittal part of the portal vein generally from its ventral aspect. There is pronounced anatomical resemblance between these venae paramediales sinistrae craniales and the cranial branches of the vena paramedialis dextra in the right half of the liver.

In 25% of the specimens the site of origin of the main branch is at the ventral surface of the sacculus. In 35%, it is between sacculus and curvature of the left portal vein trunk. In 25% (28% according to Cournaud), at the cranial pole of the sacculus ventralis and in 5% at the ventral surface of the curvature. This vessel (or after its division into branches these vessels) runs (run) in a ventro cranial direction.

In 35% of the specimens an additional branch is seen arising from the medial surface of the sagittal part of the portal vein on the cranial side of the vena paramedialis sinistra caudalis. It curves ventrally and cranially into this segment. In 4% of the cases it arises straight from the concave side of the bend running in a cranio ventral direction.

Generally, one at least of the terminal branches of the venae paramediales sinistrae caudales is also directed cranially.

As far as we can judge (for, to give exact percentages would necessitate analysing every liver), arterial supply often takes place from the left paramedian artery, though there are some times supplied from the arteria caudalis lobi sinistri.

Bile is drained into the ductus paramedialis sinister, but we have found on several occasions that the bile duct runs straight to the adjacent duct formed ventrally to the *curvatura trunci sinistri venae portae* from the junction of ductus caudalis and ductus cranialis lobi sinistri.

Before proceeding to discuss the variations of the Glissonian system in the right half of the liver, we should mark it plain that the division of the right paramedian lobe into a cranial and caudal area is an artificial one serving only to facilitate a systematic treatment of this matter.

Lobus paramedialis dexter Cranial area

Portal vein Apart from some minor individual variations, impossible to describe separately, this area is in general supplied by two to four major branches from the dorsal fun, and, by the same number of major branches, from the ventral fun, their direction is cranior, cranio lateral and cranio medial.

Hepatic artery The arterial branches, which accompany the venous branches of the dorsal fun in this area arise in 80% of the cases from the arteria paramedialis dextra and in 18% from the arteria dextra, in 2% from the arteria hepatica dextra. As all arterial branches having a common course with the portal vein branches of the ventral fun arise from the arteria paramedialis dextra, we were able to compare our data with the percentages found by Herley *et al* in a large number of cases (70 crabs). These authors found in 87% of their specimens that the cranior area of the right paramedian lobe was supplied by the arteria paramedialis dextra and in 10% by the arteria dextra. In one of their cases, the latter artery arises from the left hepatic artery.

OF THE GLISSONIAN SYSTEM

Bile ducts The bile ducts accompanying the portal vein branches of the dorsal fin in this area empty in 78% of specimens into the ductus paramedialis dexter in 18%, one or more branches open into the ductus dexter and in 4%, one or more branches drain into the right hepatic or common hepatic duct. The bile ducts corresponding with the portal vein branches of the ventral fin empty in 98% of the specimens into the ductus paramedialis dexter and in 2%, into the right hepatic duct.

Lobus paramedialis dexter (caudal area)

Portal vein The branches of the dorsal as well as of the ventral fin run in caudal, lateral and medial directions.

Hepatic artery The arterial branches corresponding with the portal vein branches of the dorsal fin prove in 72% of the cases to originate from the arteria paramedialis dextra, in 22% from the arteria dextra and in 6% from the arteria hepatica dextra. The arterial branches accompanying the branches of the ventral fin all arise from the arteria paramedialis dextra. Herley *et al* found that the arterial supply of this segment was via the arteria paramedialis dextra in 86% of their cases and in 14% via the arteria dextra.

Bile ducts The bile ducts corresponding in this area with the portal vein branches of the dorsal fin empty in 72% of the specimens into the ductus paramedialis dexter in 16% one or more branches from this area open into the ductus dexter and in 12% one or more branches drain into the ductus hepaticus. These figures are for the branches of the ventral fin 98%, 2% and 0%.

Lobus dexter

The vena dextra bends in a caudo lateral direction with a curvature ranging between 90° and 120°. A major branch the venacramialis lobidextra arises right at the curvature running in a cranio lateral direction. The terminal part of the vena

dextra (vena caudalis lobi dextri) gives off a number of vessels in a horizontal direction parallel to one another (venae marginales), their average number is four. A variable number of vessels in cranial or caudal direction, the venae intermediales, may be present between the origin of the vena paramedialis dextra and the curvature of the lateral vein. The usual number is only one, however. The vena intermedialis, adjacent to the right wall of the fossa cystica, has been named by us vena paracystica.

Segmentum craniale lobi dextri

(The figures between brackets denote the percentages found by Healey, Schroy & Sorensen)

Hepatic arteries In 86% of the specimens the cranio lateral artery arises from the arteria lobi dextri (86%), in 6% (12%) from the arteria paramedialis dextra in 6% (2%) directly from the arteria hepatica dextra, and in 2% from the arteria hepatica sinistra.

Bile ducts In 100% of the cases of Healey *et al* this area drains into the lateral duct. This is not confirmed in our specimens. In 88% of our cases drainage takes place into the ductus lobi dextri in 6% into the ductus paramedialis dexter, in 2% into the ductus hepaticus dexter and in 4% into the ductus hepaticus sinister.

Segmentum caudalis lobi dextri

(The figures between brackets are those of Healey *et al*)

Hepatic arteries In the majority of cases, namely 92% (77%) the segmental artery arises from the arteria lobi dextri, in 0% (20%) it originates from the arteria paramedialis dextra and in 8% (3%) from the right hepatic artery.

Bile ducts In 86% (86%) of the cases this segment drains into the ductus lobi dextri, in 8% (10%) into the ductus paramedialis dexter, in 4% (4%) into the right hepatic duct and in 2% into the common hepatic duct.

While we are well aware that the foregoing by no means covers all the variations liable to occur we do believe we have enumerated the most significant among them

Of the more erratic variations we might cite that described by Kehr (1912) *ii*, that of a duct which he saw coming from the right half of the liver draining into the gall bladder or a more recent case reported by Norman (1950) of a bile duct deriving from the right half of the liver emptying into the ductus cysticus

If we had to generalise we might say that as a rule Nature repeats herself though the term freak of Nature witnesses to the fact that she is capable of some queer vagaries

Chapter V

THE HEPATIC VEIN SYSTEM AND ITS VARIATIONS

Up to the present, scant reference has been made to the second system of intrahepatic structures, which is composed of the hepatic veins. The arrangement of this system of veins is far less complicated than that of the Glissonian system while the structures, themselves, are subject to comparatively few, mostly minor variations. We shall therefore devote this chapter to a consideration of these structures and the variations most commonly encountered.

As was noted previously, Glisson (1654) was the first to relate the ramifications of the portal and hepatic veins in the proper way, which appears from his two illustrations (figs 4A and 4B p 10 and 11).

There are three major veins—initially called the ‘*truncus primarius venae hepatis*’ (Glisson) but later renamed the *venae hepaticae*—and several minor hepatic veins, all of which enter the inferior vena cava. It will be necessary to have a clear picture in our minds of the location of the inferior vena cava in relation to the liver before we proceed to discuss the *venae hepaticae*, on account of their close connection with that vein.

The inferior vena cava is located in a more or less deep groove at the dorso-cranial surface of the liver *i.e.* the *sulcus* or *fossa pro venae cavae* situated in between the lobus caudatus on its left and the right liver lobe on its right. The vessel here runs in the bare liver area—in between the two

leaves of the triangular ligament namely the ligamentum hepato-diaphragmaticum on the ventral and the ligamentum hepato-renaie on the dorsal side. A dorsal ligament the ligamentum venae cavae holds the vein in the fossa. If the ventral liver surface is turned cranially and the right liver lobe then rotated towards the mid line the inferior vena cava presents itself against the dorsal wall of the abdominal cavity to the right and alongside the vertebral column showing through the covering layer of mesentery the so-called caval mesentery. On incision of the caval mesentery along this vein the vessel comes into view (fig. 110c).

The groove of this vein is clearly visible upon inspection of the corrosion specimens of which the Glissonian system alone has been injected. Three openings are, moreover seen at the cranial pole of the cyst (see figs. 12, 13 and 14), corresponding with the three major hepatic veins, which leave the organ at these points. All three are situated in the plane of the fissura dorsalis. These sites can also be visualized extrahepatically in fresh livers after incision of the falciform ligament at the point where this structure merges into the triangular and coronary ligaments (fig. 80). This second hepatic gateway will prove to be as important to hepatic surgery as the porta hepatis!

The three rami leaving the organ at this eccentrically located porta : have been named vena hepatica dextra, media and sinistra. Next to these major hepatic veins a number of minor hepatic veins enter directly into the inferior vena cava becoming visible after the incision of the caval mesentery. These vessels named by Rex ventrales kleines Astwerk coming from the region of the dorsal surface of the liver will be termed by us *venae hepaticae breves*.

Of the *major hepatic veins* the vena hepatica media appears to leave the organ slightly to the left of the fissura media. For this reason we initially thought that the course of the vessel was also to the left of this fissure (Gans 1954) but subsequently discovered that this is not so.

After accidental injection of the portal vein under a higher pressure than usual the main branches of the major hepatic veins were filled with plastic probably via the sinusoids. We were highly surprised to see that these major branches which were united at the cranial pole by some plas-

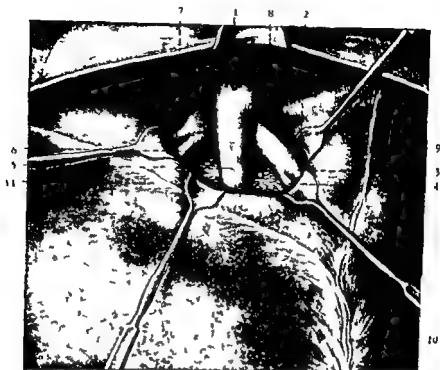


Fig 80 Specimen of upper pole of the liver ventral aspect
 1 vena cava inferior 2 venula hepatica sinistra cranialis 3 vena hepatica sinistra 4 venula hepatica sinistra interlobaris 5 vena hepatica media 6 vena hepatica dextra 7 venula hepatica dextra cranialis 8 underside of diaphragmatic arch 9 ligamentum coronarium 10 ligamentum falciforme 11 ligamentum hepato-diaphragmaticum

tic that could not escape from the inferior vena cava were lying right in the sagittal fissures. This was later confirmed in other casts.

From these observations it transpired that the three major vessels of the hepatic vein system, or one of their rami, are located in the fissures described previously, dividing the flow bed of the intrahepatic Glissonian system into a number of lobes. *The main branches of the hepatic vein system take an interlobar course!* But the main branches of the *venae hepaticae* are situated not only in the sagittally placed interlobar fissures (viz. the fissura interlobaris dextra and sinistra and the fissura medii), for at the same time the proximal parts of these three

vein trunks lie in the fissura dorsalis. Hence in the cranial section of the liver these vessels lie along the lines on which the interlobar fissures intersect the fissura dorsalis.

We are therefore able to state definitely that the fissure observed and described by Ruge in one of his fresh livers (1913) (see fig. 81) really corresponds with the right interlobar fissure, because he defined it thus: 'Die Vena Hepatica verlässt ihn (the right lateral liver lobe of Ruge) am Dorsalende der Substanzbrücke (between the *rechter Seiten*" and *rechter Strahlrippen*), sie liegt hier frei zutage

The hepatic vein, located in the left interlobar fissure, is seldom the vena hepatica sinistra itself, frequently it happens to be a side branch of the left hepatic vein.

Rex had already noted that the vena hepatica sinistra drained the left liver lobe and the vena hepatica media the two territories which he indicated as 'Astigsfolge der Ramus Ascendens' (corresponding with our lobus paramedialis ventralis dexter) and his *rechten Astwerk*' (corresponding with the lobus paramedialis ventralis sinister). The vena hepatica dextra was said to drain the *Verastlungsgebiete der Ramus Ascendens und Ramus Descendens* (corresponding with our lobus dexter) and *rechtseitiger Abschnitt des Ramus Ascendens* which corresponds with the area dorso-lateral to the fissura intermedia; this, again, corresponding with part of the dorsal fan area.

He pointed out moreover that the proximal part of the left hepatic vein unites with the proximal part of the middle hepatic vein as a short truncus communis before entering the inferior vena cava at the lateral or left ventro-lateral wall. Though this is the usual occurrence we met instances where the two vessels enter the inferior vena cava side by side.

Both Hyrtl (1873) who averred that the right hepatic vein would enter the inferior vena cava at a more cranially located point than the left hepatic vein and Rex stating the opposite are substantially wrong because the two vessels as a rule enter the vena cava at the same level.



Fig. 81. Liver with externally visible fissures. Cranial aspect.

1. Right interlobar fissure. 2. Left interlobar fissure. 3. Paramedial fissure on the right. 4. Paramedial fissure on the left. 5. Inferior vena cava. 6. Superior vena cava. 7. Superior vena cava. 8. Inferior vena cava. (after Kuge, 1913)

The *left hepatic vein* usually runs straight across the left liver lobe. It receives a branch of variable size from the left interlobar fissure, running ventrally to the pars sagittalis vene portae. This branch, the *venula hepatica interlobaris sinistra*, enters the left hepatic vein at the site where the latter leaves the left liver lobe to take its course towards the inferior vena cava. Except for some small branches, practically no blood from the left paramedian lobe is discharged into the left hepatic vein. A number of large venous branches enter the lateral wall of the left hepatic vein, but it is a striking fact that practically no vessels enter the medial wall of this vessel in the area of the left lobe (fig. 82).

The most cranial branch to the left hepatic vein, the *venula hepatica cranialis sinistra*, runs parallel to the cranial left lobar margin, usually entering the left hepatic vein within the left liver lobe. Occasionally, however, both enter the inferior vena cava as a short truncus and in one instance we even saw them entering the inferior vena cava separately.

A branch located more caudally to the left hepatic vein is situated right in the fissura intersegmentalis lobi sinistri; it is the *venula hepatica medialis sinistra*, receiving blood from both segments.

To the right of the left interlobar fissure, the left hepatic vein does not traverse the area of the lobus paramedialis sinistra, but follows a course in between the left paramedian and crusdate lobe. In this region, after incision of the coronary ligaments, the vein can be dissected out at the cranial surface of the liver (fig. 80).

Although the main branch of the *vena hepatica media* runs in the fissura media, at the site where this vessel leaves the centrocranial liver pole, it is located slightly to the left of this fissure. As it enters the inferior vena cava, either united with the proximal part of the left hepatic vein as truncus communis, or as a distinct separate branch, the middle hepatic vein can be dissected out; it can, moreover, be followed for a short distance in a retrograde direction before its disappearance into the liver.

Applying this technique we were able to inject the hepatic venous branches individually especially if the inferior vena cava had been cut too short during the removal of the organ at autopsy. This of course was contrary to our normal practice which is to inject all these branches at the same time by way of the inferior vena cava.



Fig 82 Specimen of hepatic veins Ventral aspect

1 Vena cava inferior 2 Vena hepatica dextra 3 vena hepatica media 4 vena hepatica sinistra 5 venula hepatica interlobaris sinistra 6 venula hepatica medialis sinistra 7 venula hepatica sinistra cranialis 8 venula hepatica dextra cranialis 9 Hepatic venule from the cranial area of the left paramedian lobe

Blood from both paramedian lobes is discharged by a number of venules into this vein, which is forked at the distal end. On rare occasions one may find that the blood of the cranial part of the left paramedian lobe is discharged by way of a separate vein into the left hepatic vein, located to the right of the left interlobar fissure (figs 82, 83, 84 and 85).

Nearly all the venulæ composing the *vena hepatica dextra*, which is located in the right interlobar fissure, enter this vein at its lateral or ventro lateral surface. In a few of our specimens only the blood from the lobus dexter is discharged by way of this vein, in other specimens a variable amount of blood is also carried off by this vein from the area of the dorsal fan of the right paramedian ventral lobe. As a rule however the amount of blood carried off from the right paramedian lobe is very small in comparison with the part which is discharged into the middle hepatic vein judged in proportion to the number of vessels. The right hepatic vein is the heaviest branch of the hepatic vein system. Contrasted with the lobus dexter which is usually the smallest Glissonian lobe a rather heavy branch, the *venula hepatica cranialis dextra*, may, like the corresponding branch to the left, enter the right hepatic vein occasionally it may enter the inferior vena cava slightly cranially to the latter but we very seldom found it to do so in our specimens.

We differentiate between two groups of branches from the *venae hepaticae breves* the first the *venae hepaticae caudatae* originating in the caudate lobe and usually as a definite distinct cranial and caudal branch entering high up the ventral or ventro medial vena cava inferior wall. The second group consists of a variable number of vessels of considerable size. As has been stated these vessels appear after incision of the caval mesentery. Two rather heavy branches come from the dorsal region of the right lobe of the liver running superficially immediately below the hepatic capsule (fig. 88) Elias & Petty (1952) described these veins in minute detail. They called the first which comes in laterally, the *vena hepatica dorso lateralis dextra* naming the other coming dextro caudally which enters the vena cava inferior caudally to the right hepatic vein the *vena hepatica dorsalis*. Both the *venae hepaticae majores* and the *venae hepaticae breves* fix the inferior vena cava in the liver groove.



- 1 Right stem of portal vein 2 Sagittal segment of left portal vein 3 Right paramedian vein
 4 Saccus 5 Left caudal paramedian vein 6 Lumbobulbar vein 7 Caudal vein of left lobe 8 Cranial
 vein of left lobe 9 Cranial vein of right lobe 10 Caudal vein of right lobe 11 Right hepatic vein
 12 Right cranial hepatic venule 13 Medial hepatic vein 14 Left interlobar hepatic venule 15 Left
 cranial hepatic venule 16 Left hepatic vein

Fig 83 Liver, ventral aspect

Whereas, normally, shunts are not detectable in a portal vein specimen between the different areas of ramification, we frequently found anastomoses between the ramifications of the hepatic veins as did also Elias & Petty (1952)

Having considered the normal topography of the hepatic

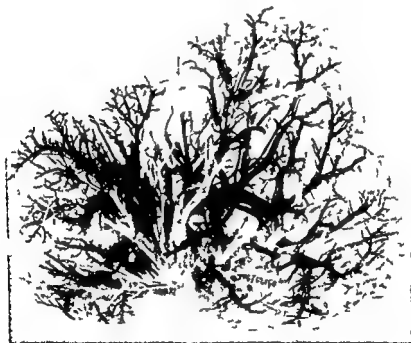


Fig 84 Heavily tinted branches portal vein Faintly tinted branches hepatic vein Ventral aspect
→ ← Portal vein branch with corresponding artery

veins let us return to the embryonic development of these veins. Contrary to Mall (1906) we imagine that when the two primary lobules meet the umbilical veins the proximal parts of these veins which are also incorporated in the expanding liver tissue retain their interlobular position unlike the distal part. Hence the vein on the right becomes the vena hepatica dextra and that on the left the vena hepatica sinistra.



Fig 8, Ventral a port Heavily united branches portal vein system Family
 united branches hepatic vein system
 → Left interlobar hepatic enule

3 4 4



2 1 5

Fig 86 Portal vein hepatic vein specimen cranial aspect
 1 vena cava inferior 2 vena hepatica sinistra 3 vena hepatica media 4 branches entering the
 medial hepatic vein 5 Right hepatic vein



Fig. 3, Portal vein hepatic vein specimen ventro-cranial aspect Maximum filling of the right hepatic vein sharply defining its area of ramification bound by line A



Fig. 100 Portal vein hepatic vein specimen with marked reduction of the left hepatic lobe.
 Dorsal a. part 1 Inferior vena cava 2 vena hepatica dorso-lateralis dextra 3 vena hepatica
 dorsalis 4 Left hepatic vein 5 Portal vein at the hilum



Fig 89 Cracked liver after injection of all finer structures Ventral aspect
 faintly united branches hepatic veins heavily united branches portal veins Complete inter-
 digitation of the structures of both systems
 1 Vena cava inferior 2 3 cm 4 Crosses 5 Unilateral incisure

After obliteration of the left proximal vitelline vein, the blood of both paramedian lobes which initially had their own efferent veins, is now discharged by way of the right proximal vitelline vein which shifts to the mid line and thus assumes an interlobular position. We agree with Mall when he says: Each interlobular vein at this stage is to form a main trunk in the adult.

In our opinion the relationship between the embryonic development of the venous system and the lobar division of the liver calls for further investigation but the subject is beyond the scope of the present study.

Chapter VI

SURVEY OF THE SUBDIVISION OF THE LIVER

In this final chapter on the anatomy of the liver, we shall endeavour to co-ordinate the details relating to the lobation of the liver set forth in the foregoing with the intention of portraying the organ in a manner that will integrate the divisions according to the Glissonian and veno-hepatic systems into a morphological and functional unity. It will be plain from what follows that we are now entering upon a highly controversial subject.

At the outset we based the internal subdivision of the human liver upon that familiar as the Glissonian system—a subdivision which, up to a point, was found to correspond to the external division initially submitted by Von Haller and later elaborated by Ruge and De Burlet *inter alia*.

Guided by the striking relationship between the two systems, we eventually mapped out an internal subdivision of the organ on the lines of the ramifications of those systems. This subdivision turns out to coincide partly with a division laid down in the past by others.

We make no claim to having uttered the last word on this complicated matter. The solutions suggested were conceived in a surgical climate—the predominant aim in the division being to resect such areas as comprise the divisional regions of the *afferent* as well as of the corresponding *efferent* structures. For all that—and we wish to make a point of stressing this before

hand—we were not able to adhere consistently to this division for all the surgical procedures dealt with in this work

Our point of departure in this division of the organ subject to the two systems was a comparison of the spatial inter relationship between the two intrahepatic systems in the lobated liver of the dog, which was initially injected to serve as a guide in our experimental surgery (see Chapter VIII), and the unlobated human liver

Somewhat closer inspection of a dog's liver will reveal certain differences as compared with the human liver. First of all there are a number of visible fissures radiating from the inferior margin at some depth towards the centre of the organ, giving the liver a lobated appearance. Secondly, it will be seen that the peripheral ventro-caudal parts of the liver are less voluminous than in the unified human liver—they consist of large flat parts in contradistinction to the centre of the organ which is far more bulky. Finally, the distance between the two hepatic gateways *i.e.*, between the porta hepatis and the point at which the hepatic veins leave the liver is relatively shorter.

There are also characteristic internal differences. Whereas the portae were seen to ramify in the human liver in all directions except dorsally, the branching of both the vena portae and of the vena hepatica is digitiform in the dog's liver. The branches of the two systems radiate like the fingers of a hand into the various lobes.

In the flat thin, peripheral areas of the liver the structures of both systems run parallel and in close proximity to each other. Towards the centre the two systems diverge *viz.*, the bile ducts with the corresponding structures of the Glissonian system towards the hilum and the hepatic veins towards the central cranial liver pole. Owing to the relatively short distance between these two gateways in the dog the distance between the different structures of the two systems is usually small yet large enough to permit the two systems to interdigitate in the bulkier central part of the liver (figs. 90 and 91)

In the periphery of the liver, however, the various areas of ramification are identical in the two systems, which accounts for the sharp demarcation of those areas by fissures and the lobated appearance thus imparted to the organ

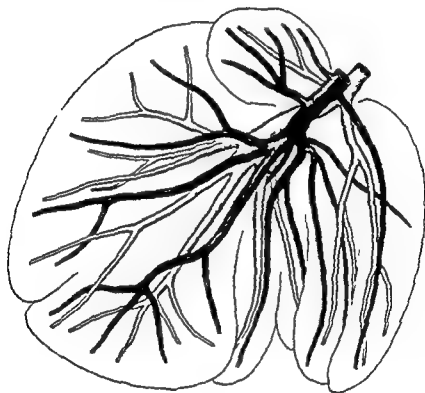


Fig 90 Liver of a dog, dorsal aspect. Light branches of the hepatic vein. Dark branches of the portal vein

Without entering into the individual structures of the two systems in the dog's liver we may generalize by saying that a characteristic difference exists in the relationship between the two systems as compared with the human liver

SURFACE OF THE SUBDIVISION OF THE LIVER 111

As we have seen, although there are indications of a lobate surface on the visceral surface of the human liver, this characteristic feature is much less pronounced than in a dog's liver. In the human organ the distance between the two gateways is relatively larger than in the dog and, in fact, the majority of mammals with lobated livers.

Another distinctive feature of the human liver is the crude lobe between the two gateways as a tissue bar, lacking in the dog's liver in which this lobe takes the form of two separate lobes turned far more laterally behind the side lobes and having no contact in the median line.

In the human liver, the branches rising from a crosswise placed vena portae situated in the sulcus transversus spread in all directions except dorsally. Although at first sight these ramifications seem to be rather chaotic, a certain amount of symmetry does appear to prevail in the origin and course of the portal main branches, as Rapp (1953) and Couinaud (1954) have pointed out. The division of the various main branches of the vena hepatica system of the human liver is digitiform in the same way as in the dog's organ.

Briefly summarising our findings for the human liver we may say that the flow beds of both the Glissonian and vena hepatica system can be subdivided into various areas. These areas are far more sharply defined in the Glissonian than in the hepatic vein system.

The main branches of the hepatic vein system are located in the interlobar fissures according to which the different Glissonian areas are demarcated and the venules entering these veins interdigitate with the portal vein ramus. There is practically no parallel course in the periphery of our specimens, such in contrast to Glisson's illustrations (see fig 4, p 10). On account of the greater distance between the two hepatic gateways the structures of both systems disperse almost immediately. Yet a correlation exists between the areas of ramification of either system. As noted previously, the area

drained by the left hepatic vein is restricted to that receiving blood by way of the portal vein bed, corresponding with the lobus sinister, while the area drained by the right hepatic vein consists almost entirely of the lobus dexter area, moreover, the middle hepatic vein drains nearly all the blood supplied via the ramifications of the portal veins of both paramedian ventral

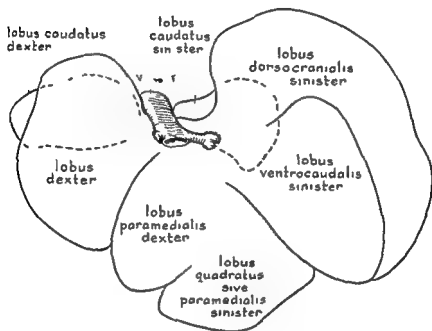


Fig 91 Division of the dog's liver into lobes ventral aspect

lobes. The caudate lobe, as was seen, has its own routes of drainage, immediately into the inferior vena cava.

Up to the present, the liver has been divided rather arbitrarily it might seem according to the Glissonian system. Is this division really an arbitrary one? After all, we have seen that this division corresponds to a certain extent with the external subdivision after Von Haller and Ruge. Gersheim & Ellis

(1954) who were obviously aware of this problem, solved it in this way. One takes the division of the portal vein branches as guides, the lobes are named after the portal branches which supply them, since among mammals the portal tree is the most constant gross anatomical system.

Judging by our own observations we believe the same can be said of the division of the hepatic veins, in the intrahepatic course of which very few variations have come to light.

In the Glissonian system of division we distinguish two halves of the liver each again subdivided into two lobes a division which as has been seen, agrees with that introduced by Flowers Cantlie etc. For the division according to the venohepatica system we can now divide the liver into three ventral lobes, a right, left and middle lobe, and this agrees with the division introduced by Ruge De Burlet and others. Thus we are here faced with a long standing controversy. This controversy, pointed out by F. Meyer (1911), was not yet understood as such because his investigation was based solely upon a study of the external aspect of the different liver types.

In between the multiple lobated liver as in the dog and the unlobated organ there exists a group of livers e.g. that of the pig the hepatic vein ramifications of which are not purely intermedial to the counterparts of the Glissonian system as existing in the unlobated type neither do they run parallel, as in the periphery of the dog's liver. It is mainly this group of livers which is probably responsible for the existing controversy, because these livers consist of three distinct lobes, though an indication of the *fissura media* is seldom lacking. The course of the hepatic veins therefore also affects the external appearance of the organ which as such may serve as an argument to those who take its intrahepatic ramifications as the basis of the subdivision of the organ.

Histologists have been preoccupied with this controversy for some considerable time but a satisfactory solution has not yet been forthcoming.

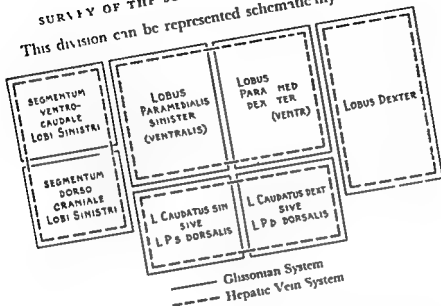
Kiernan (1833) described the lobule as a *morphological unit*, centred around the central vein (hepatic vein branch). Sabourin (1864) argued that for the liver, as a gland with external secretion, the morphological division consequently ought to be abandoned and replaced by a *functional unit*, centred around the bile duct. Bichat (1801) must have known this argument, for he remarked that nature never would have created an organ of this size for the sole purpose of producing a secretion of which the quantity stands far behind that of urine. Since Claude Bernard (1877) pointed out that the organ derives its main importance from its endocrine function, the division proposed by Sabourin has been completely discarded.

Again and again voices were raised against Kiernan's concept demanding a revision, as by Mall (1906), who introduced the 'portal unit' and recently by Rappaport *et al* (1954) who introduced the 'hepatic artery unit', both approaching the concept of a functional unit as originally proposed by Sabourin.

Similarly, one is able to divide the gross structures of the organ in three ways: firstly, a division based upon the segmental distribution of the functional unit structures (structures of the Glissonian system), secondly, the division of the organ into areas of ramification of the morphological unit structures (hepatic veins) and thirdly, a division based upon the distribution of both functional and morphological unit structures (both intrahepatic systems).

For our purpose, *i.e.*, the resection of parts of the liver we should, if reasonably possible, adopt the third alternative subdivision, seeing that a liver lobe, according to this system, not only has its own Glissonian pedicle at the porta hepatis but at the same time its own venous hepatic pedicle at the upper pole of the organ. That lobe is therefore not only a morphological or functional unit of the liver, but a surgical one as well.

This division can be represented schematically as follows



for the Glissonian system, namely the lobus dextro vesicularis (our lobus paramedialis dexter), 1 lobus praeportalis (our lobus paramedialis sinister) and 1 lobus centralis sinister (our segmentum ventro caudale lobi sinistri) The last one definitely belongs to the left liver lobe for all three types of division. However, the fact that these investigators at that time approximated the true facts solely on the basis of 1 comparative anatomical study of the external aspect of the organ, only increases our admiration for their work.

Chapter VII

INDICATIONS FOR HEPATIC SURGERY

Before proceeding to discuss the surgical implications of the foregoing anatomical findings, we would do well to consider whether or not there exists any need for surgical techniques of the kind

After studying the literature and the indications valid for classical hepatic surgery we find that the diseases of the liver which may call for surgical intervention are so numerous that it will be necessary to set a limit even to the establishment of indications. For the time being, therefore we shall consider as indicated for the new form of hepatic surgery only those solitary pathological conditions which are apparently inaccessible to any other type of therapy, leading, if untreated to the death of the patient

There are to our mind, two reasons which make this limitation obligatory firstly because this new form of hepatic surgery is still in its infancy and, secondly because this surgical therapy is a form of radical surgery comparable to a radical mastectomy an amputation and a pulmonary resection which like these procedures should be resorted to only under dire necessity

In a study of this kind, personal preference must inevitably colour a selection from the list of pathological conditions which will therefore no doubt incur criticism from some readers as being incomplete. It is nevertheless our avowed inten

tion to dwell only on the repercussions which this new form of surgery may have on the treatment of *tumours* and *deep lacerations* of the liver, *i.e.*, conditions which demand an operation as the one and only means, more often than not, of saving the patient's life. Until more experience has been gained in the new type of surgical treatment we cannot tell whether it is applicable to other pathological conditions as well.

A NEOPLASMS OF THE LIVER

It is safe to say that, as a rule, the smaller primary hepatic tumours are not diagnosed, being discovered only at autopsy. It is the larger tumours which cause symptoms and only a minority of these are accessible to the knife, depending on localisation, type of tumour and the stage reached at the time of the diagnosis.

The benign tumours qualifying for surgical exploration and, possibly, resection, are solitary tumours manifested as an upper abdominal mass, usually accompanied by mechanical disturbances. The appropriate malignant tumours for this treatment are massive, solitary tumours, revealing themselves in the same way as the benign growths, with or without the vague signs and symptoms often associated with malignant tumours, such as prostration, anorexia, loss of weight, weakness, gastric disturbances, constipation, abdominal pressure, dyspnoea, occasional pain, etc.

Because of their rather infrequent occurrence in the Western Hemisphere, solitary hepatic tumours are usually mistaken for other conditions. The differential diagnosis is in general incomplete, due to the fact that little is known of these neoplasms and operation is therefore often postponed until the condition has become inoperable. Moreover, owing to the unsatisfactory method of hepatic haemostasis and the problems arising from cholecystitis, only few surgeons attempt eradication of a primary or secondary tumour, even

though this procedure may prolong or even save the patient's life.

However, the new type of surgery, which enables the surgeon to resect liver portions along relatively avascular and aductal planes (the interlobar fissures, previously described), evades these dangers and will consequently stimulate a renewed interest in hepatic pathology. We shall therefore briefly survey some pathological features of those hepatic tumours which are amenable to surgical therapy.

The following classification of primary hepatic tumours is followed (after WARREN, 1945)

- 1) Hepatomas
 - a) liver cell adenomas
 - b) liver cell carcinomas with or without cirrhosis
- 2) Cholangiomas
 - a) adenomas of intrahepatic bile ducts solid or cystic
 - b) carcinoma cholangio cellulare
- 3) Cholangio-hepatomas of both liver cell and duct elements
- 4) Primary tumours of the liver without specific hepatic elements (vascular, fibrous, adrenal rests etc.)

Hepato cellular adenoma, which is indistinguishable macroscopically in its multiple form from nodular hyperplasia in cirrhosis of the liver, usually occurs *solitarily*. These solitary adenomata are prone to assume enormous dimensions, either because of their gradual enlargement or because of their very rapid expansion in a comparatively short period, a period liable to vary from a few months to many years.

Hepato cellular adenomata are always encapsulated, solitary tumours marked off in colour from the surrounding liver tissue. They are usually light brown or a pale grey on account of the high fat and glycogen content of the cells; sometimes these tumours are tinted by bile. Coming upon them accidentally during laparotomy the surgeon might easily mistake them for metastases in the liver.

On examining this tumour the histologist will generally find that although the cells are somewhat larger and coarser than

the liver cell, they nevertheless strongly resemble it and that the adenoma cells likewise present a trabecular arrangement

As Warvi observed, adenomata are isolated both anatomically (by a capsule) and physiologically from the normal liver tissue on account of the absence of the portal triads in the tumour, hence the bile, which is sometimes produced by the cells of the tumour, cannot be drained

These, then, are the characteristic features distinguishing this growth from the lump seen in nodular hyperplasia, which is not encapsulated (Simmonds, 1884) and in which the bile ducts do communicate with the rest of the organ

Adenomata are liable to become so large that they give the patient mechanical discomfort. It is of supreme importance to ascertain the rapidity of growth in view of possible malignant changes. For, with reference to this hepatocellular type of tumour, Deelman, Kaufmann and many others have stated that there is no essential distinction between the comparatively benign (adenoma) and the atypical malignant forms (carcinoma)

In the benign hepatocellular type of tumour, the solitary adenoma, the veins are not invaded but the benign character is due chiefly to encapsulation and transitional forms to adenocarcinoma occur in which there is invasion of veins and multiplication of tumours (Ewing). It is not surprising therefore to learn of cases in which the original diagnosis of adenoma had to be revised after its surgical removal upon subsequent metastazation (Von Bergmann, 1887, Wendel, 1911, Viradi 1937, etc.)

If the encapsulated tumour is detected during exploration, a test excision can serve no useful purpose, on the contrary, it may be injurious to the patient, because in the case of adenoma only multiple sections are capable of ruling out malignant changes. Accordingly, resection without preliminary biopsy is then indicated.

We agree with Seneque and Aurousseau (1952) that, even

if the tumour is well encapsulated, it should be resected with a generous margin and *not enucleated*, for, in the event of malignant changes, such as invasion of the vessels by the cells of the tumour, manipulation is only too likely to provoke an embolism of the tumour cell.

Though some may disagree, we share WARREN'S view that the mistaken identification of adenoma as carcinoma appears less serious in view of the mild surgical risk of exploration, which is justified in view of the rapid fatal prognosis for untreated carcinoma.

There are two distinct types of *hepato cellular carcinoma*, viz., with and without cirrhosis of the liver. Macroscopically we differentiate between three forms: solid carcinoma, multiple growths without a distinct primary tumour and the diffusely growing carcinoma.

The great majority of the hepato cellular types of malignant tumour develop in cirrhosis hepatis, a disease contraindicating resection. It is a tumour of very frequent incidence in tropical and subtropical regions populated by backward peoples. BONNE (1935) found that all his Javanese cases of hepatic tumours were hepato cellular cancers in cirrhotic livers. BERMAN'S statement (1950) that cirrhosis of the liver is always present is only related to his material. In collective statistics of 893 cases of primary carcinoma of the liver the incidence of cirrhosis was 67.2% (BERMAN).

Of the tumours occurring in livers free from cirrhosis, which, again, can be subdivided into the multiple nodular type and the solitary type, it is the latter which stands the best chance of cure by surgery, despite its rapid growth, because the rest of the liver is usually normal.

This type of tumour, which is amenable to surgical treatment, is known under the names of 'adenoma malignum' (RIBBERT, 1909, CATHALA, 1923), 'cancer massif' (HANOT & GILBERT, 1888), 'primary massive encapsulated carcinoma' (WARREN, 1945), or 'carcinoma in the form of a single

massive growth" (Nicholls *et al*, 1c Warr) Its exact incidence is unknown It may grow out at any time during foetal (Noegenrath's case, 1854) and postnatal life

According to Warr, this type of tumour frequently appears to arise from an adenoma, "and the lesion exhibits all degrees of change from benign adenoma with slight cancerous transformation to highly anaplastic carcinoma

These gray, yellow or bile stained tumours, which are encapsulated and/or project over the liver surface, may reach enormous dimensions They may be bossed in appearance, while the hepatic capsule overlying the tumour is generally thickened, and, as in the case illustrated in fig 93, firm adhesions may be present between the capsule and the adjacent organs or diaphragm In cross section the tumour may be found to be confined to one lobe (fig 94), while its colour varies from white to gray, yellow, or brownish red, the consistency also varies greatly, depending upon the extent of the degenerative changes In the tumour represented in fig 93 the cross section was lobulated

Histologically, the tumour cells may present various stages of dedifferentiation (fig 95) In the more benign forms, these cells usually resemble liver cells more or less, containing a basophile protoplasm They are arranged in trabecular formations In the more malignant forms, the cells are arranged in alveolar like formations and show little or hardly any resemblance to liver cells Even the highly malignant tumours may produce bile, however

The tumour may increase considerably in size in a relatively short period of time In Eggel's series of 14 cases (1901) exhibiting an hepatocellular carcinoma of the massive type the duration of symptoms ranged from 2 weeks to 6 months The average age incidence of this type of tumour is the 5th to 6th decade of life even though some cancers of infancy and early childhood of Dancs (1922) and Steiner (1938) collective reports belong to this group (fig 96)



Fig 93 Solid solitary primary carcinoma of left half of liver removed surgically (left hemi hepatectomy) Weight of the tumour 1950 grams size $19 \times 14 \times 12\frac{1}{2}$ cm (Operation by Pernet & Gans July 1954)



Fig. 14 Same tumour as in fig. 13 in cross section

Many authors have noted that males are predominantly affected

This group of solitary tumours is usually of unicentric origin and is therefore suitable for surgical treatment Several features

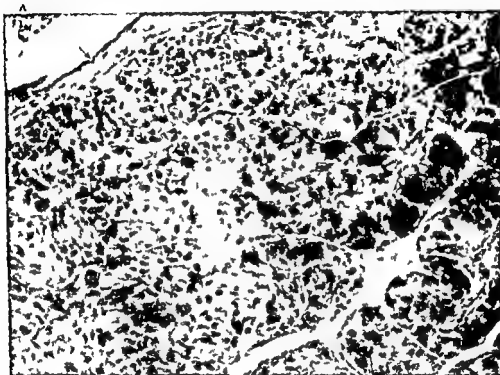


Fig 9. Microscopist's report of the tumour in fig 9.4 hepato-cellular carcinoma a capsule of the tumour

of this malignant type of tumour are still incompletely understood to day The tendency of hepato cellular cancers to invade the venous channels, in contrast to the preference of cholangio-cellular cancers for the lymph channels, is one of the problems (Herben, 1948, Seneque & Arousseau 1950 etc), another is the fact that the tumour is usually situated in the right liver lobe

Generally speaking *extrahepatic* dissemination takes place late in the course of the disease. *Intrahepatic* dissemination is quite common. Numerous small nodules, apparently metastatic deposits, may be present in the adjacent liver tissue even early in the course of the disease.

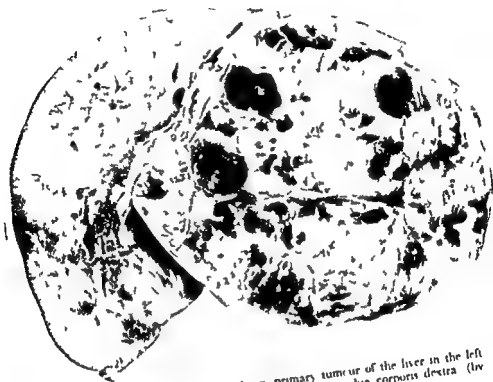


Fig 96 Encapsulated massive solitary primary tumour of the liver in the left hepatic lobe. Female 10 months with *neurohypertrophia corporis dextra* (liv. courtesy of Dr J C Hette)

X-ray treatment may give brief symptomatic relief in hepatic malignancy, as recently shown by Philipps *et al* (1954), but no lasting cure.

It will be obvious from this brief survey that these solitary hepatocellular tumours offer an absolute indication for resection.

Confidence has for long been placed in the surgical treatment of the disease, as is clear from many reports in the literature, but, owing to the risk attached to the surgical procedure itself, the high hopes entertained have in the ultimate seldom been realised, notwithstanding some spectacular results such as those obtained by Wendel (1911), Brunschwig (1947, 1953) etc

There are some other lesions which may occur in the liver at any age of life. They may either give rise to mechanical distress on account of their size, or be discovered incidentally during, let us say, laparotomy, when they may be mistaken for primary or metastatic carcinoma of the liver. Such tumours include the second group distinguished by Warvi, viz the *cholangiomas*.

We need only touch briefly on the *cholangiomas* which are congenital tumours of the bile duct epithelium, as they are far less frequent than the hepatomas. Seldom solid, they are usually cystic through the accumulation of mucus, which causes them to grow to an immense size. These tumours, which never produce bile, usually cause trouble at an earlier age than do adenomas. They may be benign or primarily malignant but degeneration to malignancy is also known though less frequently than in the case of hepatocellular adenoma. Degeneration of a cystic *cholangioma* has for all that, been reported (Willis 1943). Cholangiocellular cancers are far more rare than hepatocellular carcinoma, they represent about 1/5 of all the primary malignant tumours of the liver.

Microscopically the tumour is characterised by its glandular structure and an abundant dense, relatively avascular stroma.

Its clinical prognosis is even more rapid than that of hepatocellular carcinoma.

The following tumours viz those primary tumours of the liver which Warvi classifies under groups 4 and 5 have only one common feature, that of probably originating in a congenital malformation of tissue.

The nature of the embryonic liver tumours or hepato-blastomas (Willis) is determined by the stage of development during foetal life in which the congenital malformation arose and the kind of tissue components influencing the growth.

If there are tissue components completely foreign to the organ (group 5 of Warvi) these heterotropic tissues may be arranged in organoid structures choristomy (Albrecht 1904). Choristomas are growths which may derive from these.

Heterotropic tissues without an organoid structure such as epidermoid epithelium bone cartilage striated muscle are called teratomas.

These types of malformation probably arise at an earlier stage of development than those described in the following paragraphs.

If heterotropic tissue is absent homologous tissue components (group 4 of Warvi) may become isolated in such a way that anatomically or physiologically they are no longer incorporated in the normal structure of the organ. If we are dealing with an abnormal mixture of several homologous tissue components this condition is usually indicated as hamartoma (Albrecht 1904) when they develop we call them hamartomas. This group comprises the cholangio-hepatomas.

Malformations caused by one homologous tissue component may be divided—though occasionally no sharp distinction can be made—into areas solely composed of one of the epithelial structures which are liable to develop into hepatoma and cholangioma and those showing a preponderance of one of the mesenchymal derivatives which are liable to develop into haemangioma and lymphangioma.

Naturally enough the terminology suffers a good deal from confusion and it was for this reason that we thought it would be useful to go briefly through Albrecht's formulation.

Some tumours removed at operation, such as those reported by Ladd & Gross (1941), Benson & Penberty (1942), Lee (1942), Patton (1948), etc., all in infants and young children were described as hamartomas, but were essentially teratomas, comprising, as they did, bone, cartilage and the like. These tumours as a group, are seldom potentially malignant and the number of reported cases of *terato carcinomas* (all in young children), namely by Skifosovsk (1896), Philipp (1908), Hippel (1910), Yamagawa (1911), Castle (1914), Sheehan (1930), McRae (1935) and Roth (1938), are few.

The case reported by Lee (1942) of a tumour weighing 4½ pounds removed from a baby of 13 months, testifies to the

fact that benign teratomas are likewise liable to assume enormous proportions. These tumours may however also be multiple and have even been known to be associated with congenital abnormalities elsewhere (Schmelling 1934).

The *hamartomatous cholangio hepatomas* of which recently a few cases were reported by Kay & Tilbert (1950) are in general roughly nodular encapsulated tumours with a light reddish tan colour. Most cases occur in infants and children. These tumours may occasionally assume considerable dimensions with diameters up to 20 cm and weights up to 2 kg.

Histologically these cholangio hepatomas are composed of rounded masses of liver cells arranged in cords and bile ducts. Both anatomically and physiologically the tumour is isolated from the surrounding normal hepatic tissue. These masses lack the orderly structure of liver lobules and do not have a central vein. They are surrounded by areas of fibrous tissue containing many bile ducts and small blood vessels (Gerding *et al.* 1951).

Malignant cholangio hepatomas are usually associated with cirrhosis in which case the tumour is not amenable to surgical treatment.

Let us in conclusion consider the cavernous *haemangioma*, a benign tumour which may on very rare occasions become malignant (Ewing 1928).

The incidence of haemangiomas becoming large enough to be detectable is small. Shumacker (1942) collected 66 reports of benign hepatic haemangiomas of which 56 cases were operated upon since then this number has increased. In Shumacker's series, recently extended by Riddler & Madding (1949) the livers were enlarged and the normal tissues were extensively replaced by masses of growth with average dimensions of 12×12 cm and an average weight of 900 grams.

Macroscopically the growths appeared as knobby cavernous cysts bulging through the surface of the organ. They were blue black in colour. Usually these tumours are sharply mark-

ed off from the parenchyma by a capsule, or project from the surface as a pedunculated mass. The diffuse form is rarer. These tumours are often observed in the left liver lobe, occasionally in association with angiomas in other organs. The smaller ones are accidental findings at autopsy, they may even disappear spontaneously from thrombosis or inflammation, only leaving some scars. When these tumours develop, they may increase slowly in size with the age of the subjects (Ewing, 1928). If they become very large, they are prone to burst, causing intraperitoneal haemorrhage (Hendrick, 1948).

The few cases described as malignant haemangio-endotheliomas ran an extremely rapid fatal course (Hasting James, 1949). The question is still open for discussion whether the occurrence of these tumours elsewhere should be regarded as metastases, or only as a manifestation of the multicentric origin of the tumours (Willis, 1940).

Rare as primary tumours of the liver are, solitary hepatic tumours amenable to surgical therapy are more infrequent still. The action called for in the case of a patient suffering from a solitary tumour of the liver must depend to some extent upon the circumstances. Both biopsy and enucleation of the tumour are contra-indicated in the event of rapid growth in a comparatively short period. The future may possibly bring a more sharply defined set of indications but, for the time being *wide resection is advisable in every patient in a good general condition who exhibits a rapidly growing hepatic tumour*.

Metastatic tumours of the liver

Virchow's rule (1888) to the effect "dass fast alle diejenigen Organe welche eine Neigung zu protoplastischer Geschwulstbildung zeigen, eine sehr geringe Neigung zu metastatischer darbieten und umgekehrt" (that almost all organs susceptible to protoplasmic growths exhibit very little tendency to harbour metastases, and vice versa) is certainly not without

relevance to tumour pathology of the liver. As will have appeared from the preceding brief review, primary liver tumours are rare in our part of the world.

Secondary infestation of the liver, however, is a quite common occurrence, either by direct extension of the tumour growth as in cancer of the gall bladder, colon and stomach, or by way of the blood channels.

In Warther's series (1948) of 20660 autopsies the incidence of primary cancer of the liver was 0.26%, that of secondary tumours 4.7%.

The incidence of liver metastases in the various types of cancers as found at autopsy (Philipps *et al*, 1954) is for cancer of the breast 50%, for cancer of the bronchus 40%, for cancer of the stomach 40%, for cancer of the colon 45% and for cancer of the oesophagus 25%.

In a given case of cancer of the liver in a man, the probabilities are 20% that it is from the stomach, 20% from the intestine, 14% from the genito urinary tract and 14% from the other digestive organs. In a female the probabilities are 20% from the breast, 18% from the intestines, 13% from other digestive organs, 16% from the uterus, 12% from the stomach and 7% from the genito urinary tract (Setterlee & Anderson).

These figures show that the majority of metastases in the liver derive from those organs which drain their blood into the liver via the portal vein.

On going through 1200 unselected autopsy reports we found that the diagnosis of malignant tumour was made 165 times, 64 times of which pertained to an organ draining its blood into the liver by way of the portal vein. Fifty of these 64 cases showed distinct metastases in the liver (78%).

Schroeder van der Kolk (1853) was the first to point out that the liver is usually the first organ involved by tumours of the gastro intestinal tract—pancreas, and gall bladder—by invasion of tumour growth into the peripheral branches of the

portal vein. This idea has recently been re introduced into oncology as "portal vein type of metastazation" by Walther (1948) and Wieberdink (1950)

Serege, and later also Wassink (1915), Copher & Dick (1928) and Mermans (1946), tried to explain the localization of the pathological process in the liver

Serege, who propounded the theory of "le double courant sanguin a l'interieur de la veine porte" (the double blood stream inside the portal vein), noted that les ondes sanguines de la grande mesenterique et de la splenique, obeissent chaque a son propre courant. Elles se juxtaposent et cheminent cote a cote, parallelement a la paroi de la veine, sans se confondre separees par une barriere liquide virtuelle qui s'oppose au melange des deux sangs. (the waves of blood in the superior mesenteric and splenic veins follow each their own course. They are juxtaposed and travel side by side, parallel to the wall of the vein, without mingling, separated by a liquid barrier as it were, which prevents the two bloods from mixing)

Organs discharging their blood into the superior mesenteric vein would metastasize to the right, those by way of the splenic and inferior mesenteric veins to the left half of the liver, because of the stream lined effect of the portal vein blood. Though these observations would appear to be probably correct in the main, they do not take into account the variations occurring at the site of entrance of the tributaries composing the portal vein. As was seen previously (page 63), both Douglass *et al* (1930) and Giffillan (1930) proved by their dissections that the number of variations in the composition of the portal vein are so many, that one cannot predict with absolute certainty the site of all secondary deposits in the liver. Therefore those (Lefevre, 1935) who performed left hepato lobectomy, for example, every patient with carcinoma of the liver (Lortat-Jacob 1932)

resection of the pars dextra hepatis for gastric metastasization on the right instead of, as one would expect with Serege and others, on the left half of the liver

Hence it has been stated previously (see page 66), *the exact localisation of metastases in the liver can seldom be confidently predicted*

Late metastases of carcinoma, following removal of the primary tumour, may occur without local recurrence, as is demonstrated by the case described by Solomon & Kreps (1950) of a patient who manifested a metastasis in the liver no less than 26 years after undergoing sigmoidectomy for adenocarcinoma!

Moreover spontaneous disappearance of macroscopic secondary growth has been reported for the liver after the removal of the primary tumour, as it has for the lungs (Fletcher, 1948). This ought to be considered as a rare occurrence.

Resection of solitary metastases in the liver together with the primary tumour has been advocated (Wangensteen 1945; Brunschwig 1947, 1953 etc.)

In many instances however, it may be exceedingly difficult to determine with absolute certainty, only by palpating the organ (especially its more bulky parts) whether one is dealing with an isolated metastasis as appears for example from Golligher's series of cases. Of his 31 patients who had died a few days after radical operations on the rectum and whose liver did not appear to be involved during the operation five proved to have deep metastases in the liver (McC. Willis). An hepatogram taken during the operation may aid in giving a conclusive answer (See Appendix II).

It is a fact that isolated metastases may occur in the liver as a solitary metastasis or occasionally as a few nodules located in one and the same area. The number of these cases may indeed be greater than is generally supposed. In 10 patients who came to autopsy with cancer of the pancreas one showed an isolated metastasis without further macroscopic evidence of dissemination while in 21 cases of cancer of the stomach 2

showed isolated hepatic metastases, while 4 other cases showed a local regional lymph node involvement next to these

Brunschwig's statement (1948) that direct extension of cancer of the stomach or transverse colon is impossible on account of the fact that Glisson's capsule "is an effective barrier to infiltration of malignant cells, and in most instances actual invasion of the growth through the capsule into the liver has not occurred" applies only to the protective role of the capsule against carcinoma of the stomach and the colon

The Glissonian capsule of the fossa cystica, located in between the gall bladder and liver, however, certainly is not such an effective barrier as witness 14 cases in our series exhibiting carcinoma of the gall bladder. Four of these patients showed only microscopic involvement of the liver without any further evidence of metastasization, three others exhibited local liver involvement with extension of the tumour into the regional lymph nodes

Considering that Tam (1940) estimated the mortality from carcinoma of the gall bladder in the United States at 6500 cases per annum, the urgency of extending the scope of surgery to this disease will be realised. We have therefore long been seeking a method by which the early cases at least of carcinoma of the gall bladder could be resected. Before describing this method, we shall discuss some features of the tumour itself

The incidence of cancer of the gall bladder is found on the total number of autopsy cases varies between 0.34% (Jones 1950) and 0.81% (Von Bercenszy *et al*, 1924)

The incidence of gall bladder carcinoma on the number of autopsies cases in which carcinoma was the cause of death varies between 2.1% (Walther, 1948), 2.8% (Illingworth 1935) 3.8% (Schornagel & Strub 1954) 4.5% (Lam 1940) and 6% (Graham 1931)

In Hyden's series (1950) this disease was the sixth in the order of frequency among the fatal cases caused by carcinoma

may follow immediately upon carcinoma of the breast.

The incidence of this disease on the number of operations on the biliary tract varies between 0.87% (Vardheim *et al* 1944) and 3% (Jankelson, 1937).

Carcinoma of the gall bladder affects women far more frequently than men. Walther found the incidence in females 7.5%, Kaufmann 8.7% and Wieberdink 8.9%.

Though reports have been published on exceptionally young patients e.g. Biering's report (1946) on a 13 year old girl the average age for carcinoma of the gall bladder was 65 years (Walther's series).

The probable relationship between carcinoma of the gall bladder and cholelithiasis was pointed out for the first time by Ikerichs (1861). The incidence of gall stones in gall bladder carcinoma ranges from 65% (Walther) to 94% (Finsterer, 1932). Stewart (1931) found 30 cases with gallstones in his series of 43 gall bladder cancer patients ($\pm 70\%$), as compared with 16.4% in the control series. The incidence of cancer of the biliary tract in the cholelithiasis cases was 3.8% and 0.4% in the controls. The difference ($3.4 \pm 0.59\%$) is highly significant.

Crump (1931) found convincing evidence of the relationship between stones in the biliary tract and advancing age, he even observed an incidence of 70% for the age groups past the seventh decade.

Walters and Snell (1940) believe that the incidence of carcinoma of the gall bladder has decreased since the introduction of cholecystectomy for cholecystitis and cholelithiasis, especially after the perfection of the radiological methods of diagnosis of these diseases.

It is therefore an accepted fact that the most efficient prophylactic measure against the development of carcinoma of the gall bladder is cholecystectomy in manifest or silent cholelithiasis. Controversy rages however between those who advocate cholecystectomy as a routine procedure and those

who believe that the mortality of cholecystectomy is as high as that of cancer on the basis of stones. The latter figure varies between 2 and 5%, the former between 0.5 and 1.5% (Samburg & Garlock, 1948, Graham, 1931). This is still a considerable difference in favour of those who defend routine surgery as a prophylactic method.

Yet, in view of the fact that in Stewart's series, for example, 16.4% of all who came to autopsy suffered from gall stones, a general effort to reduce incidence of gall bladder carcinoma would imply prophylactic cholecystectomy in one sixth of the population. Because carcinoma of the gall bladder is mainly found in ageing women, we therefore believe with Graham (1931) and Schornagel & Straub (1954) that cholecystectomy should be considered in all (long standing) cases of cholelithiasis over the age of forty.

After this brief discussion of the relationship of gallstones to carcinoma of the gall bladder, we must now deal with some pathological features of cancer of the gall bladder.

The sites of predilection of cancer of the gall bladder are, in order of frequency, the fundus (54%), the neck (19%) and the wall adjacent to the liver (27%) (Vidheim *et al*). However, it is often impossible to determine the site of origin particularly in advanced cases. Carcinoma located in the two last mentioned places, in particular, invade the liver almost at once. At autopsy the whole picture may resemble massive carcinomatosis of the liver.

Irrespective of the type, carcinoma originates either in the superficial or in the deep epithelium of the gall bladder. According to Fwing it first appears as a papillomatous proliferation and induration or in eroded ulcer. That papillomas may themselves have carcinomatous properties is evident from the three cases in which malignant changes were observed by Vidheim *et al*.

There is a definite correlation between the macroscopical

appearance of the tumours and their histological composition. In the cases with a preponderance of tumour stroma (scirrhous) the tumours usually appear as firm, stony, shrunken masses, with remnants of the gall bladder contracted over the stones.

The other type of tumour presents the exact opposite, i.e. a large bulky gall bladder usually adhering to the adjacent tissues either by adhesions or by fistulous tracts. It is soft in consistency and mostly dilated. Microscopically it is usually a medullary carcinoma because of the preponderance of cellular over stromal elements.

The first type of tumour characterized as a shrunken mass is of a firm occasionally stony hard consistency. Histologically this tumour usually proves to be an adenocarcinoma or a carcinoma à cellules indépendantes.

Due to metaplasia portions of squamous cell carcinoma may be present next to the gland like tissue. It predominates only in a small number of cases. According to Gray and Sharpe (1941) plasmocellular carcinoma accounts for about 4.5% of gall bladder carcinomas removed surgically.

The second type of tumour soft and bulky in character is of a rubbery soft consistency and is white on the cut surface. The lumen is generally filled with a putrefied mass due to necrosis usually associated with infection. Perforation is therefore a quite common occurrence taking place either in the cystic duct, the liver, the anterior abdominal wall, the duodenum, stomach or transverse colon (Riedel 1911).

Histologically this tumour usually proves to be a carcinoma solidum (alveolare), an adenocarcinoma or a carcinoma à cellules indépendantes. In the last mentioned case typical signet ring cells are often found, these being large cells containing mucus with an eccentric nucleus.

A papillary carcinoma extends into the lumen and does not invade the wall of the gall bladder until much later (Cooper 1920). It grows slowly and metastasizes at an advanced stage of the disease. Necrosis may cause

haemorrhage. Furthermore the growth may block the cystic duct. Then as the result of the retention of blood and necrotic material in the gall bladder the tumour acquires a soft and bulky aspect.

Von Hanseman and Broders introduced a "tumour index", which can be regarded as supplementary to the above classification. Webber (1927), who adopted Broders' method of classification for tumours of the gall bladder, expressed the degree of anaplasia of the tumour parenchyma in microscopical sections of the tumour on a scale of I to IV.

In his series of cases Grade I and Grade II represent tumours which are only rarely of the bulky type, out of 14 cases only two palpable tumours belong to this group which contained no more than four instances of extension. On the other hand 50% of tumours of Grade III and IV showed metastases, while 12 out of 16 cases presented enlarged palpable gall bladders (predominantly of the medullary type).

The grading of tumours is a personal affair. If done by the same pathologist, however, this grading may be a valuable aid in the choice of treatment and in the forecasting of the prognosis. Vadheim *et al.* observed that the incidence of metastases increased with advancing anaplasia. In Grade I 25%, Grade II 64%, Grade III 88% and Grade IV 100% of cases showed metastazation.

Hence, the conclusion to be drawn from the statistical evidence at our disposal is that especially those tumours which appear as firm stony shrunken masses (scirrhous) when originating in the fundus or the wall of the gall bladder, can be regarded as a more favourable condition for surgical therapy than the bulky type (medullary carcinoma). Except where the development of the carcinoma concerned is papillary, this group of medullary tumours undoubtedly has an adverse prognosis.

The first organ usually involved in cancer of the gall bladder is the liver. Pryor (1908) stressed the fact that, in most of the

early cases without microscopically visible extension into the liver, there is nevertheless microscopic involvement. Vidheim *et al.* reported microscopical direct extension into the liver in 67.5% of his cases. Before starting in operation on the liver it is necessary to know which part of the organ is to be removed. That is why we studied the extent of the invasion of gall bladder carcinoma into the liver.

As noted previously (page 16), we found in our polyvinylite casts direct anastomosis between the subserosal venous channel of the gall bladder and the minor intrahepatic portal branches on either side of the fissura medii. The former structures filled in retrograde motion via the latter, after injection of the portal vein and previous ligation of the cystic vein. In addition to direct extension of the tumour, there may be haematogenous spread to the liver via these channels. When studying similar preparations of livers invaded by the direct extension of the tumour we observed that the affected area did not fill. All structures in these regions were completely occluded, either due to invasion or to external pressure. Only the major arterial branches of these areas were pressed towards the margin of the growth. Figures 97 and 98 demonstrate that the whole invaded area fails to fill. This region corresponds with the caudal regions of both paramedian lobes and part of the right lobe (see Ch. VIII).

If, with the introduction of new surgical procedures for carcinoma of the gall bladder, the possibility of this disease in the future is kept in mind, a number of cases still overlooked at present will no longer escape diagnosis. In Sainburg & Garlock's series the diagnosis was pre-operatively established in 23% of cases, though from Riedel's paper (1911) we gained the impression that this author achieved a much higher percentage with possibility of the disease in mind. At present even in relatively early cases the patient is usually inoperable by the method available, namely cholecystectomy. The progress of the disease, if the liver is involved, may definitely be

hastened by this procedure, as pointed out by Blalock (1924)

Aiga (1935) collected 10 cases reported in the literature up to 1935 of patients who had been well for periods ranging between several months and some years after the operation, and he added the case of one who had undergone cholecystec-

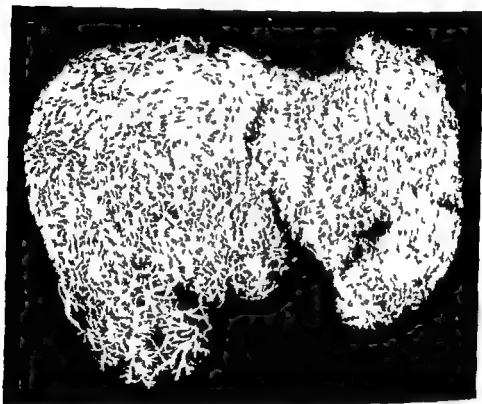


Fig 97 Gross specimen ventro-cranial aspect. Notch caused by direct extension of carcinoma of the gall bladder

tomy for carcinoma more than 7 years prior to the appearance of his paper and who was still in good condition at that time. This is an exceptionally rare case, however. Suma & William (1947) reported on 47 cases of carcinoma of the gall bladder; 23 were operated upon; cholecystectomy was carried out in 6. Only 2 patients were alive at the time when their paper was

written, 3 and 6 years, respectively following cholecystectomy. In both cases cancer was unsuspected at the time of operation the true condition being only revealed by microscopic examination.

The above facts in general confirm the statement made by

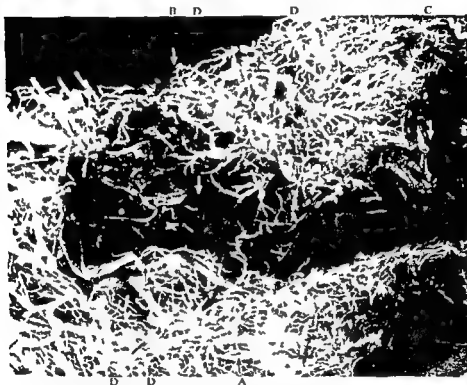


Fig 98 Dorso-caudal aspect of the corrosion specimen in figure 97. Cavity caused by direct extension of carcinoma of the gall bladder. A ventral side. B dorsal side of the liver. C proper hepatic artery. D arterial stems situated on the border between tumour and liver tissue.

Kehr (1913) the pioneer of gall bladder surgery when discussing his series of 2000 operations on the biliary system 179 of these operations were for carcinoma and only 2 of these cases were cured und das waren Zufallsheilungen denn ich operierte nicht wegen eines Karzinoms sondern wegen eines

Empyem der Gallenblase und find erst das Karzinom nachdem die Gallenblase aufgeschnitten war' ("and those were only chance cures, seeing that I was operating, not for carcinoma, but for empyema of the gall bladder, and did not discover the cancer until the gall bladder had been excised ")

In some of the reported cases of carcinoma, Riedel (1911) even questions the diagnosis on the ground that "das Mikroskop getauscht hat und chronisch entzündete Gewebe mit Karzinomatosen verwechselt sind" ("the microscopist had been misled into identifying chronic inflammation of the tissue as a carcinomatous condition") In certain cases, such as proliferating glandular cholecystitis, it is undoubtedly difficult to differentiate with confidence between inflammation and a malignant growth

Only very few surgeons of the past ventured on a more extensive procedure as originally proposed by Pylr (1908), namely, the removal *in toto* of the gall bladder with a wedge shaped part of the adjacent liver tissue Of recent years only a few isolated reports have appeared from Thorek (1947) Sheinfeld (1947) and Masse & Dubourg (1950), all attacking the liver in this old fashioned way

In Chapter X we shall describe how, on the basis of the anatomical findings in corrosion specimens of livers invaded by cancer of the gall bladder described in the present chapter, we decided on a radical operation in which the gall bladder is removed *in toto* with both paramedian lobes of the liver

II TRAUMATIC INJURIES OF THE LIVER

'In ancient times surgeons built up their experience in the workshops of war and war throughout the ages seems always to have served as a stimulus to advancement in surgical knowledge and craft' (Gordon Taylor, 1912) And it is indeed a fact that the battlefield surgeon sees a large contingent of liver wounds There are, for all that, many other causes of liver injuries In former times the kick of a horse was a notorious

one but at present, due to the increasing mechanization of traffic car accidents account for a great number of these injuries. They may be due to compression, collision or crushing by the wheels, bumpers and steering wheel. The over all risk of exposure to trauma has become greater in our time of ever increasing mechanization not only of transport but also in industry and agriculture. Falls from a more or less great height are a relatively common cause. Stab wounds, shell and gun shot wounds either accidental or due to direct assault are occasionally encountered.

Rupture of the liver in newborn children has been reported by Arden (1946), these infants were over mature and over sized.

Hepatic rupture occasionally occurs in labour (Burton Brown *et al*, 1949) in association with toxæmia of pregnancy. In this condition there is probably a predisposition to trauma.

The incidence of trauma in civilian life—though increasing—constitutes only a minor fraction of the traumatic injuries of brutal modern warfare.

In battlefield surgery its incidence is fairly high because of the large size of the organ. Wallace's series shows a proportion of 16.8% of all the abdominal wounds. For Liek's series (1915) this figure is 16.4% or 0.3% of all war casualties. However, these figures are probably under estimations because they only refer to the casualties who survived transport from battlefield to hospital and who were operated upon.

In war surgery we are generally dealing with penetrating percutaneous or perforating injuries, while the subcutaneous internal or non penetrating injuries are more common in civilian practice.

The subcutaneous liver injuries form a small percentage of patients admitted to the traumatic services of hospital. In the series of Wright *et al* (1947) the incidence varied between 1 and 1.25%. The age incidence in the Mikal *et al* series varied between 10 and 40 years.

Elder (1887), in his study of 365 cases of subcutaneous injury of the solid viscera, reported involvement of the liver in 189 cases (52%) and of spleen, kidney and pancreas in 1,6 cases. In Wright's series, liver and spleen are injured in about the same number of cases, together they constitute 72% of all intra abdominal injuries.

Both types, the subcutaneous and the penetrating injuries, will be described separately in a short discussion of the morbid anatomical features.

Subcutaneous injuries The friability and the position of the liver combine to render this organ liable to direct and contrecoup injuries. In Mikul's series, the pars dextra hepatis was lacerated nearly five times more than the left lobe, and in Wright's series, even thirteen times. The larger and more laterally situated pars dextra is evidently more easily injured.

In contrast to the opinion of some authors who think the cranial surface of the liver the most susceptible part, in Mikul's series both the ventral and ventro cranial as well as the dorsal surfaces of the liver were lacerated with equal frequency.

Laceration of the convex surface in a sagittal plane is probably the type most frequently encountered.

The idea that most ruptures take place along the dorso-caudal surface where the liver is pressed against the spine (O'Neill, 1934) is disproved by Wright *et al*, among others.

Rupture of the liver may occur transcapsularly or subcapsularly. In the latter event the capsule is intact, in the former it is also ruptured. In the first case ramponading of the centrally or subcapsularly situated laceration will occur. This division into trans- and subcapsular does not take into account size and depth of the wound or the degree of involvement of total tissue, factors which as stressed by Martin (1947) are important for the prognosis.

Hæmatoma is formed in subcapsular lacerations but the

hemorrhage is tamponaded by the capsule and/or the adjacent hepatic tissue. This is subsequently followed by abscess or cyst formation.

The liver when injured transcapsularly shows a tendency to splitting and cracking in a stellate fashion usually accompanied by hemorrhage and choleorrhagia. This bleeding is generally profuse and often tends to be persistent. Wright *et al* summarized the causes of persistent bleeding, namely the friable liver tissue does not act as a tampon. The diaphragmatic movements make the injury worse. The spilled bile retards clotting and promotes infection. And finally the thin walled intral hepatic veins do not possess valves and do not retract or contract.

Our perfusion and injection studies showed that small lacerations already cause a massive leakage from the portal vein bed. In deeply lacerated livers, leakage occurs from both portal vein bed and, to a somewhat lesser extent from the hepatic arterial branches. The bleeding from the hepatic veins is only profuse during expiration as we observed during operation.

In both subcapsular and deep transcapsular lacerations interference with the arterial and venous blood supply results in multiple infarctions, focal necrosis and sequestrations are common sequelae. The surface of a fresh wound of the liver is ragged and blood stained. Within 24 hours it assumes a dirty yellow appearance as the result of local necrosis. Later on it becomes bright yellow due to staining with bile. (Hamilton Bailly 1941)

In *percutaneous*, penetrating or perforating injuries of the liver the lacerations are usually commensurate with the size and the shape of the missile. As regards gunshot injuries there is considerable controversy on the relationship between the distance from which the gun was fired and the extent of the damage (Liek). It is a certain fact that the shorter the distance

the greater the damage by both missile and explosion. The whole organ can even be shattered by gunshot fired from nearby, but that the explosion is not alone responsible for the the damage done is evident from a case described by Ilek (1915) in which the shot was fired from an infantry rifle at a distance of 80 metres, shattering the organ irreparably. Complete fracture of some parts may occur, a considerable part may be detached, especially from the anterior edge, and may either lie free in the abdominal cavity or be carried away entirely from the body of the liver. Fractured ribs and in driven ribs frequently cause serious hepatic trauma in addition to the injuries caused by missiles. The greater the distance from which the shot is fired, the smoother is the channel bored and the fewer are the fissures radiating from it. An hepatic abscess may develop around a retained missile, this ought to be removed if possible.

Penetrating as well as subcutaneous injuries may be accompanied by *associated lesions* in other organs. In perforating injuries other organs, such as the right kidney, adjacent viscera, pleural cavity, and lungs, may also be perforated, depending on the site of entry and the direction of the tract of the missile. But as Busing (1920) demonstrated in two cases, it may be extremely difficult to determine the extent of the damage, even if both port of entry and port of exit are present. This author advised exploration of the whole abdomen for perforation. In 27 cases of perforating injuries Ilek found 11 times associated injury to the right and once to the left lung, once to the right kidney and once to the pancreas.

In Mikul's series of cases with *subcutaneous* liver injury, the kidneys, spleen and lungs were involved in 20%, 10% and 17% respectively.

We found the following statistics for the two World Wars. In the First about 75% of the hepatic injuries were uncomplicated. The mortality of uncomplicated liver wounds (in World War II) was about 10%, if one additional viscus was

injured, 28% and if two other organs were injured, nearly 40% (De Bakey, 1917)

The *symptomatology* of both the subcutaneous and the perforating injuries depends on the extent of the damage to the liver. Though the latter is always followed by haemorrhage, shock is not present in all cases. The blood that has escaped and occasionally the bile will, however, give rise to a peritoneal reaction. Slight oozing causes some physical distress, with pain over the right hypochondrium radiating to the right or left shoulder (Rehn's sign) and with accelerated breathing.

In the event of shock (as in 80% of Mikals' cases on admission) free fluid may be demonstrable in the abdominal cavity. The abdomen becomes distended, usually accompanied by a rise of temperature and accelerated pulse, pallor, restlessness and weakness. These symptoms may however occur after a lucid interval varying between some hours and some days, in which case we are usually dealing with a secondary haemorrhage. Biliary colics, haematemesis and melaena may form part of the clinical picture (Rudstrom, 1951; Epstein *et al*, 1952). Dullness in the right flank is an almost constant feature (Burnet *et al*, 1950).

Bradycardia reported by some investigators (Finsterer, 1910 and others) is not a common occurrence according to other authors (Wright and others). Occasionally a palpable mass is found which proves to be a subcapsular haematoma or a distended gall bladder, filled with blood. If jaundice follows it does not become manifest until 48—72 hours after the injury.

In all cases with a history of blunt trauma or penetrating injuries the chest should be carefully inspected for collapse of the lung due to pneumothorax.

Abdominal puncture, if performed, must be done in all four quadrants (Wright *et al*). X-ray examination may reveal an elevated diaphragm on the right side.

Secondary manifestations may follow the initial symptoms

Complications such as sub phrenic abscess, cholelithiasis, etc will be discussed in chapter XI. At present we shall deal with only that form of hepatic failure which is known as hepato renal syndrome.

The first published case of death from renal insufficiency after liver trauma is probably that reported by Furtwängler (1927). Death ensued three days after the injury, an extensive diffuse epithelial degeneration was found in the convoluted tubules of both kidneys at autopsy.

The symptomatology of the hepato renal syndrome is characterized by extreme hyperpyrexia, azotemia, oliguria collapse with cold and moist cyanotic skin, hypotension, rapid thready pulse and delirium. The urine usually contains erythrocytes, casts and varying amounts of albumen, anuria may follow, in which case the alkaline reserve is progressively depleted, leading to a lowered carbon dioxide combining power.

Mason *et al* (1925) observed that small pieces of liver, cut loose and put back into the dog's abdomen, rapidly caused a fatal toxemia. The associated symptoms point to serious injury to the liver and kidneys. A few grammes were sufficient to bring about death within 18—25 hours. The same happened when small bits of liver tissue were inserted into the chest or axilla, peritonitis thus being excluded.

MacDonald *et al* (1928) demonstrated that the liver protein was the responsible factor for these symptoms. This liver protein is therefore believed to act as a toxic substance affecting both liver and kidneys (Helwig, Boyce).

The normal detoxifying function of a lacerated liver is impaired both by the trauma and the toxic action of the devitalized liver tissue, while the renal damage is probably due to an increase of its normal detoxifying duties, which are increased by failure of the detoxifying function of the liver (Boyce). *Removal of all devitalized liver tissue is therefore definitely indicated.* According to De Brakey (1947), 85% of deaths after

liver injury in World War II were due to associated renal failure

Not all untreated traumatic lacerations of the liver are fatal, as appears from a case reported by Fabricius Hildanus (early part 17th century) of a young man stabbed during a quarrel who suffered from a severe haemorrhage. A large piece of liver later appeared in the wound and was removed by forceps. The patient nevertheless recovered (Schroeder, 1906).

With the accumulated experience of Mayer, Otis, Fidler, etc., however, the conviction has been gaining ground that immediate laparotomy with control of haemorrhage is imperative in the majority of traumatic liver cases.

Ludwig Mayer, who published the first report on hepatic trauma in 1872, found a mortality of 59% in the 276 cases which he collected. Otis (1876) found a mortality of 62% in 181 cases observed during the American Civil War. Edler (1887) collected 104 cases and in another series of 543 cases the mortality was 66.8%. The mortality figures in these and in some recent series of cases treated without operation are: Burnet *et al.* 57.1%, Wright *et al.* 75-96%, Mikal *et al.* 81.8%. That is why Mikal again recently advised operating immediately and controlling haemorrhage with the aid of gel foam, oxidized cellulose, etc., because of the high mortality in these series of cases treated conservatively.

The first case of traumatic liver injury successfully treated with control of haemorrhage was published by Von Burckhardt in 1887. From this time onwards surgical treatment has increasingly been accepted as indicated in these injuries.

In 1896 Ferner & Auvray reported the first series of 56 surgically treated cases of traumatic liver injury, adding another 42 cases in 1901. The mortality was about 30%, following suturing or tamponade of the laceration; occasionally resections were carried out.

As far back as 1910 Boljarsky observed an increase in the

mortality rate in proportion to the interval between the injury and its surgical treatment, consisting of *tamponade to control haemorrhage*. Operation within 2-3 hours resulted in a mortality of 15%, after 4-5 hours of 40%, after 9-10 hours of 66.6%, after 12-14 hours of 83%, etc. In other words, the longer the delay before exploration, the more adverse the prognosis for the patient. In Mikal's series, surgery within the first three hours after admission led to a mortality of 33.3% rising after three hours to 66%. All his patients appeared to be out of shock when operated upon.

Sometimes the symptoms attending lacerations of the liver may be very confusing, an early diagnosis being impossible. In these cases, however, it is being more and more accepted that exploration should be carried out. "If we do not explore the patient, we may deprive him of his chance to live" (Furchild, 1931).

Hence agreement prevails as to whether one should or should not operate.

Although the statistical evidence quoted shows that immediate laparotomy is indicated for this traumatic condition, mortality is still high, as Mikal *et al* has been able to show. This in our opinion is due to the procedure adopted for controlling the haemorrhage, *i.e.* tamponade with or without suturing of the laceration.

Considering the difficulties and risks to be faced during and after resection of parts of the liver until recently, this conservatism in surgical treatment is not surprising. For all that resection is indicated in the event of deep lacerations.

As early as 1920 Wendel remarked that there is no sense at all in tamponading or suturing the laceration so long as the surgeon has no information whatsoever about the vitality of the hepatic tissues located distally to the laceration. Interruption of the blood supply to this area will result in gangrene and necrosis of the devitalized parts of the liver, as rightly pointed out by this author. "Man setzt den Tampon auf ein

Gewebe, über dessen Lebensfähigkeit man nichts weiss, nur um die Blutung zu stillen. Nicht viel anders ist es bei schweren Rupturen mit der Naht. Es kann keinem Zweifel unterliegen dass nach beiden Richtungen hin—Blutstillung und Zurücklassen nekrotisierenden zum Infektion geneigten Lebergewebes—die Leberlappenresektion viel günstigere Verhältnisse gibt (We tamponade a tissue, quite in the dark as to its viability for the sole purpose of controlling the haemorrhage. And it is much the same with serious ruptures and a suture. There can be no doubt at all but that resection of the hepatic lobes is far more satisfactory, both as concerns haemorrhage control and retention of necrotic readily infected liver tissue.) (Wendel, 1920) This is all the more admirable as sound thinking in those days inasmuch as tamponade with or without suturing are generally accepted procedures to-day and are moreover constantly being advocated in current literature (Martin 1947 Mikal *et al* 1950, Scott 1951 etc.) We feel we must therefore emphasize the imperative necessity for resection in deep lacerations, and submit that it is false medical thinking to depend solely upon antibiotics and blood transfusions for the prevention of complications such as haemorrhage, hepatorenal syndrome biliary peritonitis and subphrenic abscess.

Chapter VIII

INTRODUCTION TO HEPATIC SURGERY

Since the introduction of surgery for hepatic pathology, most of the attention has been centred upon the problems involved in the control of haemorrhage. Tinker (1905) stated that oozing is apt to be profuse and persistent with ordinary cutting methods. The liver tissue is friable and frequently does not hold ligatures or sutures particularly well. Large vessels embedded in the liver substance are sometimes difficult to pick up with artery forceps to tie readily. The many methods of controlling haemorrhage which have been used indicate that there is no single infallible method.

We shall briefly discuss the various methods adopted in the past for the resection of sections of the liver and the attempts made to control haemorrhage.

Removal of pathology with the aid of *thermocautery* (Escher 1880) soon proved to be harmful because of the danger of secondary bleeding after demarcation of the eschar, the frequently insufficient control of haemorrhage and the fact that this coagulated tissue may invite infection (fig 99).

Garre (1907) rejected thermocautery because of its incompetence to control haemorrhage from the larger sized vessels while the charred liver surface hindered the surgeon from grasping the individual veins.

Radiocutting has been considered less destructive than actual

curetry because it cuts much faster, however it is hardly efficacious in arresting oozing and worthless in severe bleeding Anschutz (1903) advocated the blunt division of the liver which recently was applied again by Quattlebaum (1953)

Elastic ligature compression of the stump is one of the oldest procedures (Couzins 1871) sometimes this tourniquet was held in place by fixation pins, while the stump was extra peritonealized This method has long since been discarded because of the slow healing and the risk of infection

Tamponade of the raw liver margins following resection as advocated by Anschutz (1903) initially with iodoform gauze (Mickulitz) soaked in warm saline to which adrenaline was added, is a method now probably practised by no one Yet in those days some surgeons came to the conclusion (e.g. Ransohoff 1904) that immediate closure of the laparotomy wound ought to be rejected unconditionally The abdomen was therefore left open over the packed liver area To tamponade gunshot tracks Schroeder (1906) even devised a rubber bag which could be inflated after its introduction into the tract of the missile

During World War II good results were attributed to the use of pieces of macerated muscle as a tampon which was applied to or sutured on the bleeding surface It was thought that a haemostatic agent was liberated from this muscle promoting arrest of the bleeding All sorts of grafts have been advocated omentum (Bojarski 1910) tendons the falciform ligament, the gall bladder (Wendel 1911) etc In all kinds of lacerations tamponade of the wound with gelatine sponge or oxidized cellulose soaked in thrombine is at present a generally accepted procedure (Scott 1951)

Rubber covered clamps, especially the Doyen clamps have been used and advocated by Garre (1907) McDill (1912) and others Initially these clamps were left on the liver for 24 to 48 hours for which reason the abdominal wound had to be left open (Clement 1890 Kocher 1902) This procedure

Chapter VIII

INTRODUCTION TO HEPATIC SURGERY

Since the introduction of surgery for hepatic pathology, most of the attention has been centred upon the problems involved in the control of haemorrhage. Tinker (1903) stated that 'oozing is apt to be profuse and persistent with ordinary cutting methods. The liver tissue is friable and frequently does not hold ligatures or sutures particularly well. Large vessels embedded in the liver substance are sometimes difficult to pick up with artery forceps to tie readily. The many methods of controlling haemorrhage which have been used indicate that there is no single infallible method'.

We shall briefly discuss the various methods adopted in the past for the resection of sections of the liver and the attempts made to control haemorrhage.

Removal of pathology with the aid of *thermocautery* (Escher 1880) soon proved to be harmful because of the danger of secondary bleeding after demarcation of the eschar, the frequently insufficient control of haemorrhage and the fact that this coagulated tissue may invite infection (fig. 99).

Garré (1907) rejected thermocautery because of its incompetence to control haemorrhage from the larger sized vessels while the charred liver surface hindered the surgeon from grasping the individual veins.

Radiocutting has been considered less destructive than actual

cintery, because it cuts much faster, however it is hardly efficacious in arresting oozing and worthless in severe bleeding. Anschütz (1903) advocated the blunt division of the liver, which recently was applied again by Quattlebaum (1933).

Elastic ligature compression of the stump is one of the oldest procedures (Cousins, 1871) sometimes this tourniquet was held in place by fixation pins while the stump was extra peritonealized. This method has long since been discarded because of the slow healing and the risk of infection.

Tamponade of the raw liver margins following resection is advocated by Anschütz (1903) initially with iodoform gauze (Vickulitz) soaked in warm saline to which adrenaline was added is a method now probably practised by no-one. Yet in those days some surgeons came to the conclusion (e.g. Ransohoff 1904) that immediate closure of the laparotomy wound ought to be rejected unconditionally. The abdomen was therefore left open over the picked liver area. To tamponade gunshot tracks Schroeder (1906) even devised a rubber bag which could be inflated after its introduction into the tract of the missile.

During World War II good results were attributed to the use of pieces of macerated muscle as a tampon which was applied to or sutured on the bleeding surface. It was thought that a haemostatic agent was liberated from this muscle promoting arrest of the bleeding. All sorts of grafts have been advocated omentum (Bohrskis, 1910) tendons the falciform ligament the gall bladder (Wendel 1911) etc. In all kinds of lacerations tamponade of the wound with gelatine sponge or oxidized cellulose soaked in thrombine is at present a generally accepted procedure (Scott 1951).

Rubber covered clamps especially the Doyen clamps have been used and advocated by Garre (1907) McDill (1912) and others. Initially these clamps were left on the liver for 24 to 48 hours for which reason the abdominal wound had to be left open (Clement 1890 Kocher 1902). This procedure

has been discarded because of the danger of infection. Though the clamps are still used, they are now taken off after suturing the raw liver margin after the resection. Thin liver edges are preferably compressed digitally.

A variety of *hepatic sutures* have been advocated, all being modifications of that favoured by Kusnetzoff and Pensky (1896). A large blunt needle was used to prevent injury to the vessels, threaded with a double piece of heavy catgut, one of the threads of which was cut after the needle had passed through the liver tissue and was then tied. After division of the liver, the large vessels were circumstitched in the cut surface.

The disadvantage of 'cutting through' of these sutures was avoided by the application of magnesium plates, introduced by Pyle & Martin (1905). These were narrow perforated plates, consisting of pure magnesium of variable length fastened along the line of resection against the ventral and dorsal liver surfaces by means of heavy catgut. When the catgut had been tied the raw liver margins were coaptated, if possible.

Wendel (1911) introduced an interlocking type of mass ligature which he applied in a resection of the right liver lobe for a primary cancer of the liver. Before resection, he ligated the right hepatic artery and right hepatic duct but refrained from ligaturing the right portal vein trunk for fear of thrombosis. The patient was well for over 9 years after the operation.

This is as far as we could trace one of the very first surgical

Fig 99 Major methods of resection

- 1 Elastic ligature and extraperitonealization of the stump (Laws, 1874)
- 2 Simple cautery (Laquelin) removal (Lacher 1886)
- 3 Ligation with reported less bleeding for physical line of cleavage used (Langenbuch 1888)
- 4 Hemostatic suture with blunt needle and closure of liver surfaces after resection (Kusnetzoff and Pensky)
- 5 Clamps (a) for hemostasis and (b) completely exteriorized (Lapoint 1897) or (c) only the handles exteriorized for removal through a stab wound (Kocher 1902)
- 6 Ligation with temporary (Anschütz 1903)
- 7 Hemostatic sutures placed proximal to clamps and electro-cautery excision of tumor distal to clamps. Raw surface closed by interrupted sutures going at and the longitudinal hemostatic sutures preventing cutting of sutures through liver tissue (Warwick *Artery of Surg Clin and Obst* 1913)

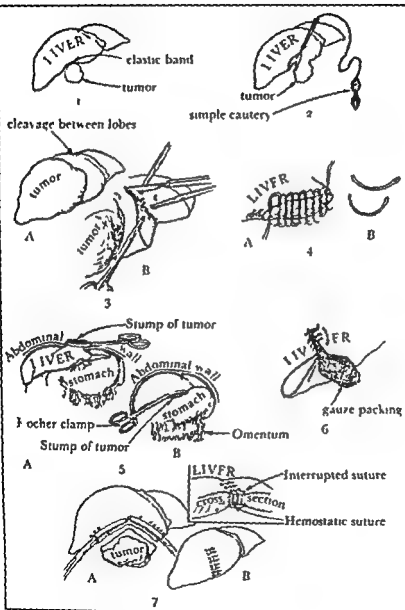


FIG. 99

procedures performed along an avascular plane of the liver through fossa cystica and fossa venae cavae with control of bleeding at the hilum, which is in sharp contrast to the bloodless form of surgery, advocated by Baron (1910), Wangensteen (1951) and others, where by the hepatic artery and portal vein were compressed during surgery in the hepato duodenal ligament

Comparatively early, both Keen (1897) and Langenbuch (1897) realized that the fossa sigttilis sinistra of Von Haller should be regarded as a relatively avascular plane, along which they resected the "right or left liver lobe"

Keen, Langenbuch and Wendel therefore have a legitimate claim to being the pioneers of an anatomically sound form of hepatic surgery. Wendel went a step further than Keen and Langenbuch, who had for long been resecting along an avascular plane, when he first ligatured the structures at the hilum

Keen's work, among these authors was generally known and adopted, in fact until quite recently, there had been little advance since Keen described his operation (1899) in which the left liver lobe was severed with a Paquelin crutery and the larger veins were closed with the tips of the finger following which the crutery was temporarily laid aside. I passed a catgut ligature under each vein by means of a Hagedorn needle, and one of my assistants tied slowly but firmly. Five ligatures were thus applied. A packing was applied which was removed after 48 hours. "No haemorrhage occurred, but bile escaped to a considerable extent from the 4th to 12th day (Keen)

In surveying all the methods advocated, one is not surprised to learn that nearly every case of liver surgery, if the patient survived has appeared in the literature. This in itself proved how risky these surgeons considered this procedure and how lucky they felt that the operation did not end fatally (Bar & Schalm 1954)

Judging from the number of cases reported it appears that around 1910 hepatic surgery enjoyed more interest than at any other time. The number of cases however gradually decreased and after 1920 reports were no more than sporadic. For this reason we have no hesitation in saying that this form of hepatic surgery never became popular even though Warren (1945) tried to re-introduce the best techniques of all the old methods. The principle of the *new form* of hepatic surgery which we are about to describe is based on a thorough knowledge of the anatomy of the organ in contrast to the various forms summarised above.

At first it was thought that the problems could be solved by technical means without taking account of the intrahepatic structures the only exceptions being the three authors mentioned viz. Keen, Ingenbush and more especially Wendel and those few who had adopted Keen's technique. But the general rule was to resect parts of the liver regardless of their relationship to the totality of the organ and without foreknowledge of injury or otherwise or ligaturing structures afferent to or efferent from distal liver areas which as a result remained behind often devitalised after the resection.

As Martin (1947) has pointed out repair is retarded when major amounts of liver tissue are devitalised until full sequestration occurs. The devitalised tissue not only increases the risk of primary haemorrhage but also of choleorrhagia and secondary haemorrhages liable to occur during sequestration. In a subsequent chapter (XII) we shall consider other complications apt to be produced by the presence of devitalised hepatic tissue viz. infection (abscesses) hepatorenal syndrome and the like.

It is owing to these facts collectively and individually that none of these techniques ever became popular. As we have seen Wendel* was the first to practise well.

* Among Wendel's references we found Thole to whose work we were not able to have access.

informed anatomically sound surgery. Cantlie (1898) already had demonstrated "that the gallbladder occupies a central position in the liver, that on either side of it lie the true right and left lobes of the liver and that a line from the fundus of the gall bladder to the exit of the hepatic veins divides the liver into equal portions", a conclusion independently confirmed by Wendel and Martens (1920).

These two authors realized that resection on one liver half should be performed along the vascular plane through gall bladder fossa and vena cava inferior, following ligation of the unilateral structures at the hilum of the liver, especially since Wendel had obtained such excellent results in a patient whom he operated upon in this way (1911) for what initially appeared as an adenoma, but 9 years later it autopsy proved to be a hepatocellular carcinoma.

As far as we have been able to trace, no surgeon ever applied this principle between 1911 and 1952, till finally this procedure was reintroduced by Seneque *et al* (1952), who performed a left hemihepatectomy for hydatid cysts.

Meanwhile, anticoagulants had become available since 1911 liberating ligation of one of the trunks of the portal vein from the risk of thrombosis. Intrahepatic mass ligation had thus become superfluous, seeing that *bleeding could be controlled from the hilum* by there ligating the unilateral structures prior to resection. Others had sought to solve the problem along similar lines.

There was Riven (1919), for instance, who had worked out an operation for the resection of the left hepatic lobe after ligating the structures to the hilum. He ligatured to the hilum the left trunk of the portal vein, the left hepatic artery and the left hepatic duct, after which he resected the left lobe of the liver by thermocautery. This procedure however, falls short of the best anatomical and physiological standards in that it leaves the left paramedian lobe devitalized.

After ligaturing the right hepatic duct, the right hepatic

artery and the right trunk of the portal vein, Fortat Jacob and Robert (1952) resected the right half of the liver together with the quadrate lobe. Although the resected parts do not correspond perfectly with the internal morphology of the organ, as Couinaud (1953) justly observed, their work attracted attention for the sound novel methods it introduced into hepatic surgery.

We, for our part, are persuaded that these authors were stimulated by the latest developments in lung surgery to adopt the procedure of ligaturing the various structures to the hilum of the liver before proceeding to resect. By the same token Couinaud, Herley and we; ourselves, inspired by the benefit accruing to modern pulmonary surgery from a thorough study of the anatomy of intrapulmonary hollow structures have pursued an analogous course for hepatic surgery.

The legitimate inference to be drawn from anatomical research in the interests of surgery is that rather than *one* there are *two* hepatic gateways, each with its own specific significance. For in addition to the hilum we have the point at the cranial pole of the liver where the hepatic veins leave the organ. As has been shown, the veins there can be dissected for some distance. The plane in which the veins lie *the dorsal fissure* (see figs 12-14 and 101-103), separates the ventral and dorsal paramedian lobes. Not only do these veins mark off the dorsal fissure, but, as we have stated before, they proceed from there to the three sagittal interlobar fissures by which the various Glissonian areas are bounded.

These fissures are ideal planes for the resection of parts of the liver firstly because incision along these planes is itself a safeguard against the notorious complications attendant upon the classical form of surgery *i.e.* the risk of haemorrhage, cholerrhagia and occlusion of those structures which are distal to the plane of resection possibly involving other very real dangers (see Chapter XI) in addition to morphological or

functional loss of hepatic tissue, secondly because, even if these planes are not always visible on the surface, the major hepatic veins, by their very position, mark them off.

These planes are avascular—except for the hepatic veins situated within them and the hepatic venules which may pass across them—and contain no portion of the bile ducts. It has been found in actual surgery that, by opening the liver along these planes, hemorrhage is avoided, provided the hepatic veins or venules be left intact. Technically, this is a very simple matter, as we had occasion to note with Bix in dogs and later with Pernet, during a hemi-hepatectomy, using the method introduced by Anschutz (1903) and recently recommended again by Seneque *et al* (1952) and Quattlebaum (1953), which consists in *incising the liver bluntly*! In this way one can be sure of not opening the hepatic veins which intersect the plane. These small vessels can be dissected, for which we used the handle of the scalpel, afterwards ligaturing them individually.

The importance of these planes warrants a closer description of them with reference to figs 12-14 and 100-103.

The *left interlobar fissure* cuts the ventral surface of the liver along a line corresponding to the connecting line between the umbilical incisure and the left wall of the caudate lobe. The plane through this fissure passes along the left lateral wall of

Figs 100-103. Cross-section specimen of a liver. All structures of Clowman system injected.

Fig 100. Ventral aspect of the liver.

Fig 101. Same specimen as fig 100. Right paramedian lobe removed. 1. Right paramedian vein. 2. Right vein. 3. Sagittal part of left stem of portal vein. 4. Left cranial paramedian vein. 5. Plane of intersection through left interlobar fissure. 6. Plane of intersection through right interlobar fissure. 7. Front view of caudate lobe. 8. Vessels of the gall bladder (injected via cystic artery and cystic vein).

Fig 102. Same specimen as fig 101. Left paramedian lobe removed. 4. Left cranial paramedian vein. 9. Sacculus. 10. Left caudal paramedian vein.

Fig 103. Same cross-section specimen as fig 102. Cranial segments of right and left lobes removed.

11. Cranial vein of left lobe. 12. Caudal vein of left lobe.

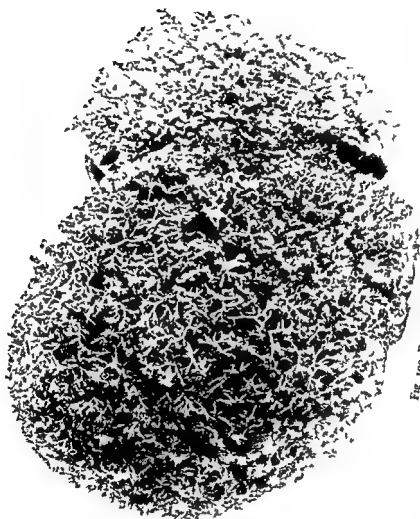


Fig 100 For legend see page 160

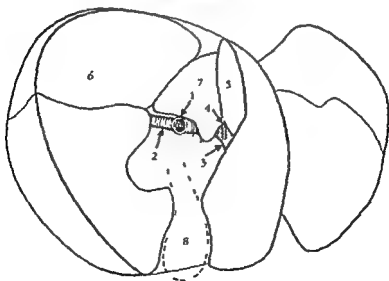
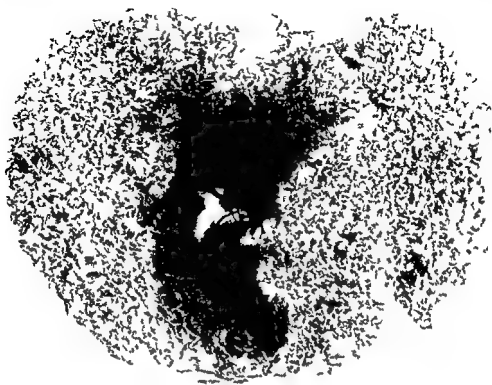


Fig 101 For legend see page 163

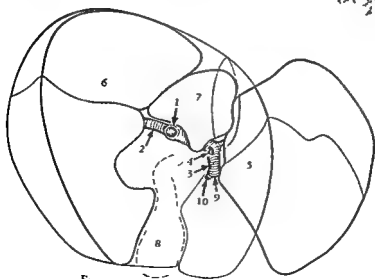


Fig 102 For legend see page 160

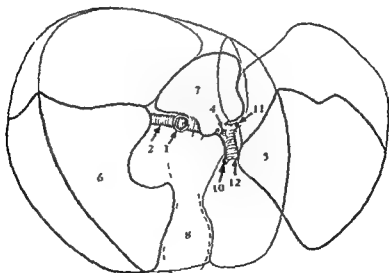
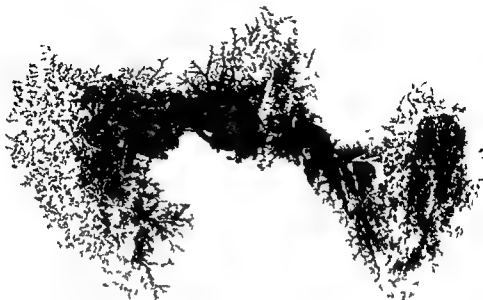


Fig 103 For legend see page 160

the inferior, hence it is not perpendicular to the horizontal plane which can be imagined as passing through the hilum, but forms an angle with it varying from 45° to 75° , with the mouth of the angle turned to the left. Thus the plane through the fissure runs roughly parallel to that through the medial fissure.

The plane of the left interlobar fissure cuts the sagittal part of the left portal vein into two halves, a medial half from which the left cranial and caudal hepatic veins arise and a lateral half from which come the cranial and caudal veins of the left lobe.

The left interlobar hepatic venule which marks off the plane of the fissure, runs ventrally to the sagittal part of the left portal vein. The calibre of this vessel is usually considerable. Any resection of areas medial to this vein (e.g. middle hepatic lobectomy or the resection of the pars dextra hepatis) should spare this vein as it almost exclusively drains blood from the left lobe of the liver (see fig. 82). In sum then the plane of this fissure can be fairly simply defined with reference to the following points: the connecting line between the umbilical incisure and left lateral wall of the caudate lobe, the left lateral wall of the venous cava inferior and the left interlobar hepatic venule.

As we have already been able to establish (Chapter VI) the left lobe of the liver is an hepatic lobe in a surgical as well as anatomical sense in that it possesses its own afferent and efferent structures. The Glissonian pedicles are found in the umbilical fossa and the hepatic vein pedicle (left hepatic vein) is between the two leaves of the coronary ligament (fig. 80 p. 94).

The medial fissure cuts the ventral liver surface along a line described by Cantlie as running from the fundus of the gall bladder to the exit of the hepatic veins. The plane of this fissure is demarcated by the middle hepatic vein, the most

proximal part of which is situated in the line on which the medial fissure and the dorsal fissure intersect, thus cutting not only the left half of the liver off from the right half, but also the ventral middle lobe from the dorsal

The middle hepatic vein drains the blood from the middle lobe, except from the dorso lateral area of the 'dorsal fin'. Hence the middle lobe is a liver lobe in both an anatomical and a surgical sense, because it has its own afferent and efferent structures at both gateways

The intermedial fissure, on the other hand, which cuts the right paramedian lobe into two parts, viz., the areas of the ventral and dorsal fin, has no surgical value. It is for this reason that we do not regard these two areas as segments, for they have neither their own segmental Glissonian pedicle, nor an intrinsic blood drainage. Hence this contrasts with the two segments of the left lobe of the liver.

The right interlobar fissure is less readily identified at the surface of the liver than the fissures referred to previously. The right hepatic vein is situated in the plane of the fissure rather more frontally than sagittally. The fissure divides the right paramedian lobe from the right lobe. Usually the fissure intersects the right lateral wall of the cystic fossa.

Before proceeding further, we would do well to consider the variability of the position of this fissure. It was stated in Chapter I that the right interlobar fissure seldom cuts the dorsal surface of the liver along the line indicated by Herley *et al* and Cournaud, but is inclined rather to follow a line which usually approaches or cuts the cystic fossa. This would account for the fact that in carcinoma of the gall bladder, the right paramedian lobe as well as the caudal segment of the right lobe is affected. The part of the right lobe which is attacked by this disease consists of a thin slab of hepatic tissue. In many cases it seems to us unnecessary not to say unavoidable to

remove the whole right lobe merely for the sake of this small section, in other words to perform a resection of the pars dextra. We have therefore made a special study of this area with the



Fig 104 Specimen of the Glissonian system dorsal aspect A fossa cystica
B venae paracysticae

help of thirty corrosion specimens the results of which will now be related

The condition we found in 60% of our specimens was as illustrated in figs 12 and 104. It will be seen that usually one, but sometimes two paracystic veins issue from the vena dextra before it bifurcates into the cranial and caudal veins of the right lobe. The area of ramification of the paracystic vein

proximal part of which is situated in the line on which the medial fissure and the dorsal fissure intersect, thus cutting not only the left half of the liver off from the right half, but also the ventral middle lobe from the dorsal

The middle hepatic vein drains the blood from the middle lobe, except from the dorso lateral area of the 'dorsal fin'. Hence the middle lobe is a liver lobe in both an anatomical and a surgical sense, because it has its own afferent and efferent structures at both gate ways

The intermedial fissure, on the other hand, which cuts the right paramedian lobe into two parts, viz., the vens of the ventral and dorsal fin, has no surgical value. It is for this reason that we do not regard these two vens as segments, for they have neither their own segmental Glissonian pedicle, nor an intrinsic blood drainage. Hence this contrasts with the two segments of the left lobe of the liver

The right interlobar fissure is less readily identified at the surface of the liver than the fissures referred to previously. The right hepatic vein is situated in the plane of the fissure rather more frontally than sagittally. The fissure divides the right paramedian lobe from the right lobe. Usually the fissure intersects the right lateral wall of the cystic fossa

Before proceeding further we would do well to consider the variability of the position of this fissure. It was stated in Chapter I that the right interlobar fissure seldom cuts the dorsal surface of the liver along the line indicated by Herley *et al* and Cournaud, but is inclined rather to follow a line which usually approaches or cuts the cystic fossa. This would account for the fact that in carcinoma of the gall bladder, the right paramedian lobe as well as the caudal segment of the right lobe is affected. The part of the right lobe which is attacked by this disease consists of a thin slab of hepatic tissue. In many cases it seems to us unnecessary, not to say inadvisable, to

corresponds to the medial area of the caudal segment of the right lobe and covers the same ground as that assigned by Herley *et al* and Cournaud to the right paramedian lobe. The paracystic veins which in this group of specimens may arise from any point situated between the origin of the right



Fig 106 Glissonian system specimen dorsal aspect. Both right and paracystic vein absent. A vena caudalis lobi dexteri B fossa cystica.

paramedian vein and bifurcation of the vena dextra—a distance which proved to vary from 1 cm to 5 cm—can be visualized either from the right incisura or along the vena dextra after the liver has been opened bluntly (see p 174).

In 40% of the specimens there was no paracystic vein at all



corresponds to the medial area of the caudal segment of the right lobe and covers the same ground as that assigned by Herley *et al* and Cournaud to the right paramedian lobe. The paracystic veins which in this group of specimens may arise from any point situated between the origin of the right



Fig 106 Glissonian system specimen dorsal aspect Both right and paracystic vein absent A vena caudalis lobi dexteri B fossa cystica.

paramedian vein and bifurcation of the vena dextra—a distance which proved to vary from 1 cm to 5 cm—can be visualized either from the right incisura or along the vena dextra after the liver has been opened bluntly (see p 174)

In 40% of the specimens there was no paracystic vein at all

and in 10% this was found to be due to the fact that the vein formed part of the dorsal fin. It was only in this group of 10% that the right interlobar fissure cut the dorsal surface of the liver as reported by Healey *et al* and Cournaud (see figs 18 19 and 105).

In 30% of the specimens the absence of the vessel was accounted for by the fact that the vena dextra was lacking. In a case of this kind, the right trunk (see fig 106) divides into three branches, one, the right paramedian vein, in a ventro caudal or ventral direction, one, the caudal vein of the right lobe, in a lateral or latero caudal direction, and the third, the cranial vein of the right lobe, in a lateral or latero cranial direction*.

In all these specimens the right interlobar fissure either cut the right wall of the cystic fossa, or ran along the lateral wall of the cystic fossa.

The surgical implications of these facts are that, prior to resection of the middle lobe for early gall bladder carcinoma, it is necessary to ascertain the course of a paracystic vein. If there is one, it should be ligatured, as the area of ramification of this vein has to be included in the resection.

Now, the boundaries of this area to be resected can seldom be established with absolute certainty beforehand. What the surgeon does know during resection, after the paracystic vein has been ligatured, is that he is working within those boundaries if incision does not provoke bleeding. But if hemorrhage does occur, it may be taken as a warning that the border of this area has been violated and in that case the best procedure is to resect the whole caudal segment of the right lobe *in toto* with the middle lobe.

In the absence of the paracystic vein, a middle lobe resection will suffice in the first stage of carcinoma of the gall bladder. If, on the other hand, the vena dextra is lacking, the alternatives are either to resect the caudal segment of the right lobe

* An insight into the anatomy of the intrahepatic portal venous system may be gained by means of a portal venogram (see Appendix II).

in *toto* with the middle lobe after ligaturing the segmental pedicle, or to resect the pars dextra in its entirety. It should furthermore be pointed out that the right lobe, like the middle and left lobes, is a lobe of the liver both anatomically and surgically, for the reason that intrinsic to it are a Glissonian pedicle and an hepatic vein stem.

The various structures of the Glissonian and of the hepatic vein systems can usually be dissected from the three lobes as well as from the two halves of the liver both at the hilum and at the upper pole of the liver. At the latter this is a fairly simple matter the interior leaves of the triangular and coronary ligaments are incised at the cranial point of transition to the falciform ligament. By dissecting straight along the dome of the liver, the vena cava inferior is reached and here the three main stems can be exposed (fig. 80). The corresponding vein allied with the hepatic lobe to be resected can be followed intrahepatically for some distance as we did with dogs with impunity.

In two of our dogs the abdomino-thoracic approach was used. After ligation of the cystic duct and cystic artery the Glissonian pedicles of the middle lobe were followed into the liver the overlying liver tissue being incised bluntly. No bleeding occurred. These structures were ligated intrahepatically at the desired place.

Then the corresponding hepatic vein was identified at the cranial pole of the liver. This main hepatic vein and several of its branches were dissected bluntly over the whole length in a caudal direction. There was some slight oozing but this had completely stopped at the time the abdomen was closed. The hepatic vein was ligated intrahepatically under direct vision at the same level where the Glissonian pedicle had been ligated beforehand. The middle lobe was removed *en bloc* with the gall bladder.

Both dogs did very well post operatively. They were sacrificed three months after the operation. The only signs of previous surgery observed at autopsy were the absence of the gall bladder and the fixation of the omentum which had been approximated to the cut surface (fig. 107). There were no signs of portal vein thrombosis and no visible traces of the extensive dissections at the hilum or in the body of the liver.



Fig. 107. Dog's liver seen ventro-cranially. A fissure in the area of the resected middle lobe. Remaining lobe thickened and with rounded margin as

It is necessary to bear in mind that the walls of these major hepatic veins are very thin and may easily be torn. They should, therefore, not be clamped (Bix, 1951). The course of the interlobar or resection plane—whichever the case may be—can be determined by the course of these veins, if not altogether, then at least part of the way.

In view of the important role played by the hilum in hepatic surgery, we think it would be appropriate to recapitulate briefly the main points of the topography of the various structures referring to Chapters III and IV for a more detailed description.

As we have seen conditions at the porta hepatis are far more intricate. Accordingly, Couinaud favours approaching the hilum, not only dorsally as he advised at first, but ventrally at need. He was induced to take this line by ‘certaines critiques (qui) ont été formulées concernant leur possibilité et leur innocuité. Il semble que le nombre des variations segmentaires ait suscité de légitimes alarmes, comment sur des faits soumis à d’assez nombreuses variantes construire une technique simple susceptible de faire face à des dispositions aussi diverses?’ (some criticism made as to their possibility and innocuousness. Apparently the number of existing segmentary variations has legitimately raised some alarm. How can a simple technique, capable of coping with such diversity, be based upon facts subject to so many variants?)

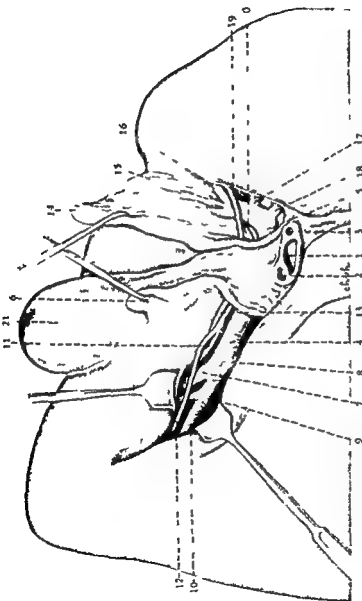
For the frontal approach to the hilum, Couinaud reverses the normal method by which the lobar structures are first picked out at the two hepatic gateways and ligated, after which the liver is incised. He first incises the liver along an interlobar fissure and ligatures the lobar Glissonian pedicle intrahepatically or he opens the fissure until the part to be resected is attached only to the Glissonian pedicle after which this is ligated. Thus, in a hemi hepatectomy, the liver is incised along the medial fissure down to the hilum and in this way *the hilum is approached from the front*. To our mind, the

drawback to this procedure is the considerable haemorrhage from the plane of resection which occurs after the hepatic venules crossing this plane have been ligated. For, as the different structures at the hilum have not yet received attention, there is a rise in the intrahepatic blood pressure in the hepatic veins. The consequent bleeding is liable to obscure the area being operated upon. For all that, there may sometimes be advantages in adopting this route in preference to the caudal hilar approach, as in those cases mentioned by Cournaud, *viz.*, tumours in the region of the hilum—a condition which we personally consider as inoperable—or adhesions of the liver to adjacent tissue.

There is, however, an alternative, which surgical treatment of dogs brought to our notice. It consists in *enlarging the hilum in the course of the branches of the portal vein*, a decided asset especially on the right side, if there is no right incisura, or only a small one (in about 20% of all livers¹). It means that a right incisura can be made by enlarging the hepatic gateway from the edge of the hilum. Given a blunt incision in the produced part of the right trunk of the portal vein and the right vein and provided penetration into the liver be not too deep, haemorrhage is virtually obviated. The hilar edges are held to one side by means of eyelid retractors (modification Desmarrès). In this way even the right vein, which runs fairly superficially under the dorsal surface of the liver, can be followed to where it divides into the segmental branches, *viz.*, the cranial and caudal veins of the right lobe.

There are practically no large or medium branches of the hepatic vein at this site, at most a few minute capillaries may be encountered. This is because the surface area around the hilum discharges blood into the *venae hepaticae breves* which run almost parallel to the right trunk or the *vena dextra* (fig. 88).

Adopting this method of enlarging the hilum one can, for instance, dissect the paracystic vein(s), should this be in



118, 108. Topography of the fundus of the liver. Prepared specimen of Clissona's capsule (6 and 14).
 0 Caudal artery of left lobe. 1 Caudal vein of left lobe. 2 Cranial vein of right lobe. 3 Cranial artery of right lobe. 4 Caudal vein of right lobe. 5 Caudal artery of right lobe. 6 Caudal vein of left lobe. 7 Caudal artery of left lobe. 8 Caudal vein of right lobe. 9 Caudal artery of right lobe. 10 Caudal vein of left lobe. 11 Caudal artery of left lobe. 12 Caudal vein of right lobe. 13 Caudal artery of right lobe. 14 Caudal vein of left lobe. 15 Caudal artery of left lobe. 16 Caudal vein of right lobe. 17 Caudal artery of right lobe. 18 Caudal vein of left lobe. 19 Caudal artery of left lobe. 20 Caudal vein of right lobe. 21 Caudal artery of right lobe.

practicable in the hilum, itself, as well as some of the other territorial structures

Portal vein Each individual lobar portal vein branch can be visualized at the liver hilum. In the left half of the hilum (umbilical fossa), even the segmental branches can be identified over a considerable distance before they disappear into the liver. However, in the majority of cases the left paramedian cranial vein, if it arises from the ventral surface of the pars siguttalis cannot be reached. Visualization of the segmental branches supplying the left liver lobe can be realized by incision of Glisson's capsule in the umbilical fossa along the dorso medial margin of the left liver lobe, till after further preparation these branches appear at the bottom of the umbilical fossa (fig. 108).

A similar course can be adopted for the preparation of the territorial branches of the left paramedian lobe, the caudal branches (left paramedian caudal branches) at the medial aspect of the recessum transversum be seen, the cranial ones are visible in only 35% of the cases (namely when they arise from the medial instead of the ventral surface of the siguttal part of the left portal vein) after proper dissection. Therefore the capsule should be incised along the dorso lateral margin of the quadrate lobe.

Disregarding the condition in which the right portal vein trunk divides at its very origin (bifurcatio venae portae), when the right paramedian vein is the direct continuation of the main portal vein trunk (20%), and the condition which was encountered in 10% of the specimens, in which the right paramedian vein originates from the left portal vein it may be said that this branch generally arises right at the margin of the right hilum. Incision of the capsule along the right hilar margin and right incisure and retraction of the overhanging liver edges at this site generally enabled us to visualize both the right paramedian and the right vein. After removal of the

enveloping connective tissue of Glisson's capsule the right hepatic artery can be followed intrahepatically for a distance of at least 1 cm. The same holds true for the venous dextral to the origin of the right portal vein.

Hepatic artery. In upper abdominal operations such as gastrectomy ligation of the aberrant hepatic or accessory hepatic artery—which if no anastomoses between this vessel and the branches of the proper hepatic are present at the hilum are endarteries for the liver areas supplied by them—may result in necrosis of these areas (lobes or segments) if no antibiotic treatment is instituted. This may explain at least some instances of liver death reported after upper abdominal surgery.

On the other hand Segall while discussing the numerous anastomoses between the intrahepatic branches of the hepatic artery and the phrenic arteries by way of the coronary ligaments and the hepatic capsule pointed out. Ligation of the hepatic proper distal to the origin of the arterial gastrica dextra when the hepatic artery is previously healthy results in more or less extensive necrotic changes and eventually necrosis is a probable outcome that must be feared. But if the artery is diseased then it is permissible to ligate at this point with freedom from fear of untoward results because one can count upon a previously developed collateral circulation. A number of hilar dissection specimens were compared with the plastic casts. These comparative studies brought to light a rule of great importance to hepatic surgery namely that the intrahepatic distribution of the different branches of the left hepatic artery can be predicted from their location at the hilum provided that their extrahepatic course has been partly visualized by dissection. It is therefore necessary to trace the artery to the site where it penetrates into the body of the liver tissue.

Immediately before entering the liver the arterial branch joins the

corresponding portal vein branch and bile duct in the umbilical fossa or left hilar area

This fact holds true for the arterial branches supplying the left liver lobe, the caudal area of the left paramedian lobe and the caudate lobe. The branches for the left lobe finally join the cranial and caudal left lobar veins at the place where these two (occasionally three if an intermedial vein is present) enter the liver, they can be visualized in the same way as previously described for the portal vein branches. The left paramedian artery, usually coursing medial to, occasionally dorsal to the pars sagittalis venae portae, can be seen disappearing into the caudal area of the left paramedian lobe after joining the corresponding vein and bile duct at the medial aspect of the saccus. Rarely, if ever, can the cranial arterial segmental branch be recognized as such.

On the right there are two anatomical features of surgical significance, viz

(1) An arterial branch, usually the arteria dextra but sometimes a segmental branch, curves around the basis of the right paramedian vein during its lateral course. An anterior relationship, in which this artery curves over the basis of the right paramedian vein, was found in 80% of the specimens while a posterior relationship, with the artery curving underneath the basis of this vein, was present in 20% of the specimens (13% in Couinaud's series).

(2) The right paramedian artery usually runs along the medial aspect of the corresponding vein, namely in 78% of our specimens (65% of Couinaud's specimens), but occasionally along the lateral surface (18%), anterior surface (2%) or posterior surface (2%) of this vein. Sometimes two (10%) or even three (2%) arteries are present running along this vein.

While dissecting the right paramedian vein from its Glissonian tissue the surgeon should beware of injuring the branch curving around the basis of this vein. Only the visible structures

are dealt with when resection of the right paramedian lobe begins. The right paramedian artery, running along the medial surface of the corresponding vein, is divided between ligatures (mounted on a Deschamps suture carrier), after which the corresponding vein is treated in the same way. By leaving the ends of the ligatures long, they can be used after the division of the vein to retract this structure out of sight. If present an arterial branch in lateral or anterior position now comes into view (pulsations!) and can be attended to. The right artery distal to the site of origin of the right paramedian artery can some times be followed for a short distance intrahaptically. Occasionally the branches of the dorsal fan may arise from this artery. Some of the arteries running laterally to the right paramedian vein belong to this group. Blunt dissection with the back of the handle of the knife is recommended for the purpose of following this artery, which is carried down to the *vena dextra*. This vein lies fairly superficially below the visceral surface of the liver.

Bile ducts The bile ducts are often difficult to visualize because of their ventral position. Usually however a distinct right and left hepatic duct can be recognized at the porta hepatis before their junction with the common hepatic ducts. The rule, mentioned previously as valid for the left hepatic arterial branches which can be applied for identification purposes during a hilum dissection also holds true for the bile ducts forming the left hepatic ducts. The ducts coming from the left lobe caudate lobe and caudal area of the left paramedian lobe can be identified in the same way as the corresponding arteries and veins. Even in those instances where the ducts cannot be visualized properly in the umbilical fossa on account of their ventral position to the veins one or two suture ligatures applied with a wide margin round the junctions of these three structures will equally take care of the duct.

It will thus be evident that, whereas the arteries and veins

are seldom overlooked during a hilar dissection, the bile ducts frequently are. We therefore do not resect the gall bladder until the end of the operation and utilize it (by pinching) for visualizing the bile ducts. In extensive hilar dissections it may moreover be advisable to dissect out the common bile ducts for a short distance and to apply a rubber covered clamp, so that no bile can escape into the duodenum. This clamp should in no way interfere with the blood flow in portal vein and proper hepatic artery.

The right paramedian duct always courses along the medial surface of the corresponding vein, though occasionally anastomoses of the dorsal fin may drain into the right duct by way of small ductuli lateral or anterior to the right paramedian vein. The bile duct from the right lobe runs underneath the base of the right paramedian vein.

In our opinion there are two strong arguments to be advanced against an extensive dissection at the hilum. One is that the variability of the Glissonian structures may be such as to require a very extensive hilar dissection to render the pattern intelligible. Let us recall a single instance to exemplify this. As previously stated in 18% of the specimens the left paramedian artery arose from the right hepatic artery. In this instance the arterial blood supply to the left paramedian lobe will be blocked if the right hepatic artery is ligated proximal to its origin e.g., in a right hemi hepatectomy. This however, will mean risk of necrosis of a lobe after removal of half of the liver which should certainly be prevented if possible. As similar morphological variations were found in the bile ducts the same applies to these structures with this difference that instead of necrosis, atrophy results from blocking of the bile ducts (see p. 200).

The second argument is that only that lymphatic and nerve tissue which belongs to the part to be resected should be destroyed. Although we have studied neither of these structures, we feel that the destruction of these tissues, which must

inevitably result from an extensive hilar dissection, should be avoided

Not an extensive hilar dissection can be circumvented by making straight for the lobar Glissonian pedicle, detaching it and ligating it at the most distal possible point

The approach to the Glissonian pedicles in the left half of the hilum is comparatively simple (see p 176), except to the pedicle of the cranial part of the left paramedian lobe which is only accessible frontally, via the left interlobar fissure

Dissection calls for more patience in the right half of the hilum. The right paramedian vein has to be released from the capsule as distally as is feasible. We explained in Chapter IV that in some cases the segments of the right lobe are supplied arterially from the right paramedian artery or drain bile into the right paramedian duct. These arteries to and the bile ducts of the right lobe arise from the artery or enter into the duct at the base of the right paramedian vein. It is for this reason as well as in view of the presence of the right artery and right duct that dissection *in situ* at the origin of the right paramedian vein is preferably avoided and the utmost distal location for manipulation is recommended.

The same applies to the pedicle of the right lobe. Again the farthest distal point is advised for dissection for which the hilum may be elongated if necessary. For we have seen that in a number of cases arteries supplying areas of the right paramedian lobe or bile ducts from this area issue from the right artery or discharge into the right duct. The farthest distal intrahepatic ligation of the pedicle of the right lobe will prevent occlusion of these arteries and bile ducts.

We see, then, that usually it is not imperative to open the liver to gain access to the Glissonian pedicles but that as in pulmonary surgery the hilum can be attended to before proceeding to resect. In the last resort, Couinaud's recent method may provide a solution but only if it is quite impossible to reach the structures from the hilum.

There is one exception, that of the pedicle of the cranial area of the left paramedian lobe. Cleavage of the liver along the left interlobar fissure is always necessary before this can be approached anteriorly.

Having, on p. 165, explained how to identify the left interlobar fissure, there now remains the right interlobar fissure to be discussed. It is a fissure of the very first importance to middle lobectomy.

It is an observed fact that the middle hepatic vein drains nearly all the blood from the two paramedian lobes. Hence, when this vein has been ligated, congestion is bound to occur in the area of the middle lobe. If ligatures have been applied around the lobar Glissonian pedicles beforehand, they can be ligated as soon as congestion is exacerbated. The plane along which the right paramedian lobe is brought into relief by the congestion against the right lobe is situated slightly to the left of the right interlobar fissure, as the right hepatic vein continues to drain blood from the area of the dorsal fan of the middle lobe (fig. 87, p. 104). By keeping somewhat to the right of the line of congestion, interference by passive bleeding, which may obscure the area of operation, can be avoided.

We refer to Chapter V for a more detailed description of the possible operative procedures which emerge from the foregoing.

Chapter IX

SURGICAL APPROACH TO THE LIVER

Before proceeding to discuss a number of resections we would do well to consider whether any useful suggestions can be made regarding incision. For, the present diagnostic resources at our disposal seldom provide us pre-operatively with those particulars of a surgical disease of the liver such as localization, dissemination and other factors which determine, not only its operability, but also the mode and path of approach to the process. This uncertainty makes it difficult to draw up a surgical plan of campaign, which has to depend upon what the surgeon finds during an *exploratory laparotomy*.

Generally speaking an incision should meet the requirement both of providing adequate exposure with minimum retraction and of permitting an alteration at will if dictated by unexpected circumstances. The latter, above all is very important and we must dwell upon it before we discuss the various incisions commonly employed for exploration of the right upper abdomen.

With possible resection in mind we sometimes try to approach an area in which with the abdominal incisions commonly used for exploration adequate working space cannot be obtained or only with the utmost difficulty. For resection of the right half of the liver, for instance such space is required in the right subdiaphragmatic area and the right flank a difficult space to approach particularly in the asthenic pa-

nient with his narrow costal arch, being, as it is encapsulated within the dome of the diaphragm on the cranial and caudal parts of the thoracic cage on the ventral, lateral and dorsal sides. That is why in many cases the incision used for exploration has to be enlarged.

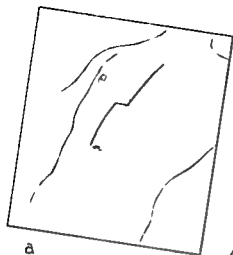
If we ask ourselves what different types of incision are generally used for exploration of the right upper abdomen, we find from an enquiry by Summers (1952) that 38.5% of surgeons use a right subcostal type of incision, 16.3% a transverse incision and 13.4% alternatively a right rectus (in the asthenic type of patient) or a transverse one (in patients of the pyknic habitus).

Every incision mentioned by Summers, except the transverse, can be used as it is, or slightly lengthened, for the *resection of the left lobe of the liver* (see fig. 111). As we ascertained in cadaver experiments, it is not even necessary to use the \perp -shaped incision recommended by Raven (1949) for this resection, nor yet the \lrcorner -shaped incision of Anschutz (1903). The incisions enumerated by Summers are less effective for *left hemihepatectomy*.

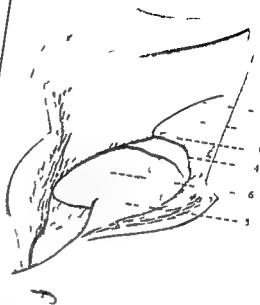
If difficulty is encountered in dissecting at the hilum or, as in the case of an asthenic patient, the cranial surface of the liver and the vena cava inferior are difficult to approach, extension of the incision by sternum splitting (see fig. 109) will be found of considerable assistance, as they were in our cadaver experiments. The skin incision is extended upwards along the axis of the sternum as far as about its centre. The xiphoid process is resected, following which the sternum is split obliquely from the base to at least one third of its length (Whitson

FIG. 109. Extension of right paramedian incision by means of the sternum splitting incision.

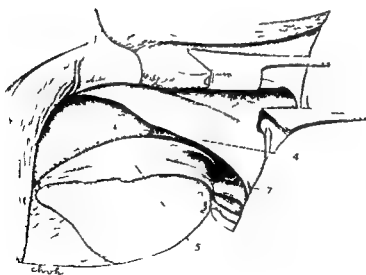
A Ventral aspect. B Xiphoid process removed. Diaphragm detached from sternum. 1 Line of incision at sternum. 2 Sternum. 3 Surface along which xiphoid process was resected. 4 Diaphragm detached from sternum. 5 Falciform ligament. C Pars dextra hepatis. D Left side view. 7 Vena cava inferior.



a



b



c

Fig 109

et al, 1954) Care should be taken to avoid opening the pleural cavity (pneumothorax) or pericardium, which are both freed bluntly from the sternum by finger dissection before the sternum is split. The splitting may be carried out by means of a pair of rib scissors. It is possible to enlarge the working space even more by dividing the diaphragm in a downward direction to the inferior vena cava.

This, however, is of no avail for operations on the right half of the liver. The problems then raised are far more intricate than those facing the surgeon who finds by exploration that a radical gastrectomy is indicated, for which Garlock (1946), Humphreys (1946), Carter (1947) and others recommend the combined abdominothoracic approach. It is for this reason that Lortat Jacob and Robert (1952) solved the problem of the approach to the right half of the liver in a similar fashion, *viz*, by extending the exploratory abdominal incision into the thoracic cavity.

This incision was developed by Rio Branco (1912). There are several modifications of this method, mainly for portocaval shunt surgery (Carter, Hervey & Humphreys, Lortat Jacob). A right rectus or right paramedian incision is used to explore the abdomen, the incision being extended upwards as far as the costal margin. When incision is to be extended into the thorax, a sandbag is placed under the patient's right side in the renal region. If resection is decided upon, a long curved incision, starting from the former one, is made over the 8th intercostal space up to the mid axillary line to the right and across the costal margin to the left. The incision is continued downwards into the chest (fig. 110 A).

The common costal cartilage is divided between the 8th and 9th ribs. A radial incision, extending from its origin at the point of division of the costal margin to the inferior vena cava, divides the diaphragm. A Finocchetto retractor is placed between the divided ribs and spread out. The falciform ligament is divided. Right lung, stomach and intestines are packed

with moist pads, the former in an upward direction the latter downwards and sideways

This procedure facilitates both mobilization of the right half and the approach to the two hepatic gateways with plenty of room Experience gained in experimental surgery has, moreover, shown that, anatomically too it is quite the best way for mobilising the liver from all sides Whether from the physiological point of view it is likewise the best approach to the organ is a debatable point There are some who feel that entering the abdomen by way of the thorax is undesirable and only a makeshift solution of the problem (Wahren & Vikgren 1954)

We know of no incision meeting all the requirements other than by way of the thorax We have tried out all existing incisions and their various modifications upon cadavers one of which only we should like to describe in more detail By cutting the costal arch (Clute and Albright 1938), more working space can be obtained in the right upper abdomen This is a principle applied by Holman (1950) in a subcostal incision parallel to the costal arch which he advocated for porto-caval shunt surgery

The procedure is to explore through a subcostal incision which is made a few centimetres below and parallel to the midcostal margin If resection is decided upon the incision is extended in a latero-caudal direction up to a point situated in the anterior axillary line at the same level as the umbilicus and medio-cranially towards the xiphoid process The lateral skin flap and the subcutaneous tissues are displaced laterally from the costal margin by dissection and a subperiosteal resection of a small piece of cartilage of the costal margin is carried out at the level of the 5th intercostal space In the broad-chested type the right half of the liver can be approached by the lateral displacement of the costal margin Excellent exposure of the hilum is obtained by means of this incision

In cadavers however we found that this incision, without opening the thorax, is only satisfactory for the broad-chested hypersthenic type of patient

Chapter X

ON HEMI-HEPATECTOMY AND SOME HEPATO-LOBECTOMIES

Possible operative procedures which emerge from the foregoing may be detailed as follows

A Hemi hepatectomies

This technique is based on the fact that the liver is a paired organ as regards its Glissonian system

The plane of division of right and left portal vein trunks, hepatic arteries and hepatic ducts and their ramifications the fissura media, divides the gall bladder bed of the liver into two and is directed cranially to the left side of the inferior vena cava. This plane corresponds to the plane of resection in a hemi hepatectomy even though the liver is not a paired organ as regards its second intrahepatic system, i.e., the hepatic veins. As described previously, this system consists of two groups of veins namely (1) the vena hepatica brevis coming from the left and right dorsal surface of the liver and each ending individually on either side of the inferior vena cava, and (2) the major hepatic veins leaving the liver at the most cranially and centrally situated area both on the right and the left side carrying the blood from the liver into the inferior vena cava just below the diaphragm. One of the hepatic veins the vena hepatica mediana, runs in the fissura media, it usually carries blood from the lobes situated on either side of the fissure to the left hepatic vein. The junction of both veins is

situated only slightly to the left of the left side of the inferior vena cava

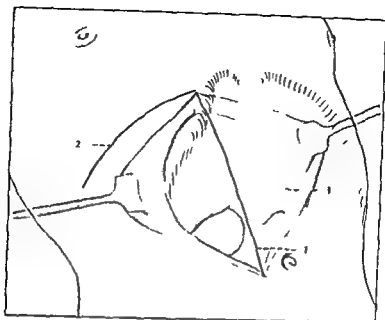
If resection of the *right half* of the liver is carried out, the liver is gently retracted downwards, stretching the triangular ligament (see fig 110 B) This ligament consists of two leaves, an anterior ligamentum hepato-diaphragmaticum and a posterior one, the ligamentum hepato-renal. Between these two leaves the cranial pole of the liver is bare and very friable. The anterior leaf is first divided the dome of the liver being followed and care taken to avoid injury to the diaphragm. To free the liver further, the hepato-renal ligament at the dorsal right side has to be severed. The right half of the liver is therefore gently rotated upwards the ligament being divided during the rotation, the scissors should remain close against the liver to avoid damage to the right adrenal gland (fig 112)

The right part of the liver is thus completely free from its surroundings and is freely movable for a considerable distance in almost all directions. When the right lobe is rotated to the left, the inferior vena cava is stretched and can be identified by its peritoneal covering. While this vessel is dissected out the venae hepaticae breves on the right become visible (fig 110 C). These veins are attended to after ligation of the hilar structures.

This is when hilar dissection may begin. The liver is rotated upwards through the diaphragmatic defect into the right lower thoracic cavity during which manoeuvre the hilar region is exposed. When removing the liver from its bed, care should be taken not to overstretch the hilar structures (fig 113).

For resection of the *left half of the liver* through an abdominal incision the left liver lobe should be turned laterally and the hilum approached from the left instead of from the right. Hence the surgeon stands at the left side of the patient (fig 111).

At the hilum the structures are ligated where the transverse fossa merges into the umbilical fossa for a left hemi-hepatectomy, and at the right hilar margin for a right hemi-hepatectomy.



a

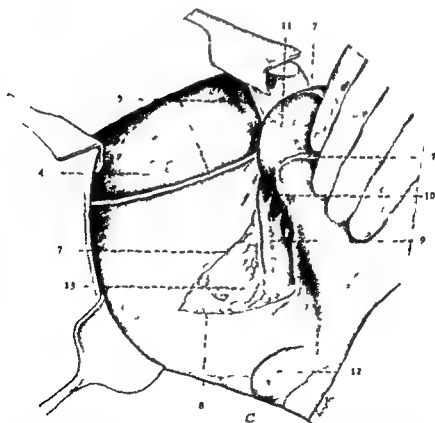
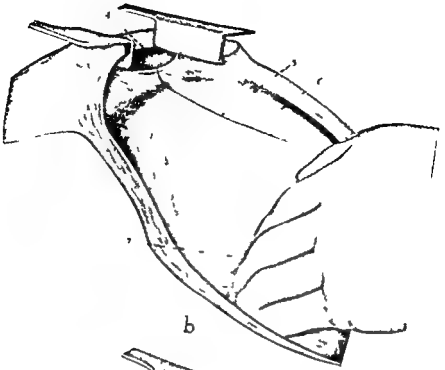
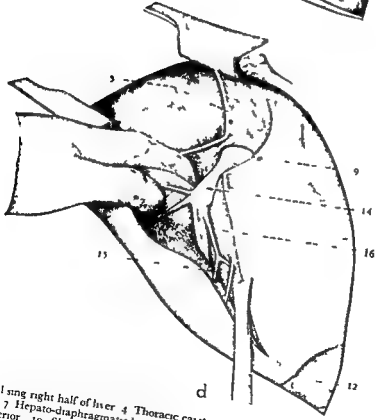


Fig 110. Right hemihepatectomy seen from the right
 A Skin incisions with exploration of abdominal cavity 1 Abdominal incision (right paramedian) 2 right rectus incision 3 Falx form ligament B Abdomen and thorax opened 4 Thoracic cavity 5 Incision of diaphragm C Diaphragmatic arch Hepatodiaphragmatic ligament stretched as result of pulling down right half of the liver



b



d

bil sing right half of liver 4 Thoracic cavity 5 Lines of incision of d a
 n 7 Hepato-diaphragmatic ligament 8 Hepato renal ligament 9 Vena
 inferior 10 Short hepatic veins 11 Bare liver area 12 Gall bladder
 ht adrenal D Separating the liver along the fissura media 5 Lines of
 of d aphragm 9 Vena cava inferior 12 Gall bladder 14 Vena hepatica
 media 15 Venulae hepaticae 16 Fissura media.

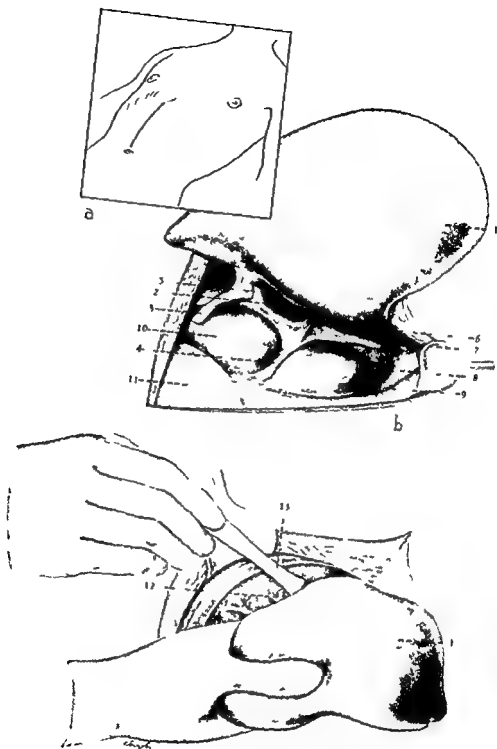


Fig. 111

c

After ligation of the Glissonian structures at the hilum of the liver, the latter will, theoretically, shrink and turn pale on account of the depletion of blood on one side. This, however, is frequently not the case, as we observed in dogs and as was noted during a hemi hepatectomy. Nor did Bix see any decoloration in his case.

Before the liver is divided, the plane of resection is marked by a sharp division of the liver capsule. Then blunt division of the liver is advisable, as performed by us first in dogs and later in a patient, its advantage is that the hepatic venules crossing the plane of resection are not opened, on the contrary, they can be dissected out, ligated and divided (fig. 110 D).

Both to prevent hemorrhages and injury to the medial hepatic vein, we advised resection of the liver along a plane parallel to and only *slightly* to the left (in a left hemi hepatectomy) or to the right (in a right hemi hepatectomy) of the fissura media so as to devitalize as little tissue as possible (Gans, 1954).

The raw liver surface is then re-peritonealized (omentum, falciform ligament, gall bladder, etc.).

B Hepato lobectomies

Left hepato lobectomy. The umbilical fossa is dissected out and the two (occasionally three) segmental branches of the left lobe are identified: the cranial vein, the intermedial vein and the caudal vein of the left lobe, the first at the convexity of the left portal vein, the latter at the saccus (fig. 111). In all our specimens the Glissonian structures joined at these points to

Fig. 111. Left hepato lobectomy.

A. Midline incision. B. Left lobe turned upwards, lesser omentum removed. Left side view.

1. Left lobe. 2. Line of incision along lesser omentum. 3. Ligamentum teres hepatis. 4. Transverse segment of left stem of the portal vein. 5. Vena caudalis lob. sinistri. 6. Vena cranialis lob. sinistri. 7. Left hepatic vein. 8. Vena cava inferior. 9. Caudate lobe. 10. Quadrate lobe. 11. Colon.

C. Resection of left lobe. Left side view.

1. Left lobe. 12. Hepatic venules. 13. Back of the handle of the knife.



Fig. 112. Division of the ligamentum hepato-renal, seen from dexter-caudally.
 1 liver 2 bare liver area 3 ligamentum hepato-renal

continue their intrahepatic course into the left liver lobe to gether. Adequate care can be taken of all Glissonian structures of the left liver lobe by wide use of transfixion sutures at the sites mentioned above, namely around these two or three structures. The dorsal surface of the liver frees the surgeon during this procedure. The liver is brought back into its normal position and held by the assistant. The left hepatic vein is ligated at the cranial liver pole. The capsule is incised sharply along the left interlobar fissure as indicated previously, after which the liver is divided bluntly along the fissure, again without opening the hepatic venules before they are ligated individually. The raw liver surface is then re-peritonealized (omentum falciform ligament, etc.)

Occasionally a bar of liver tissue bridges the space between the left caudal segment and the quadrate lobe overlying the saccus in the caudal part of the umbilical fossa. This bridge of tissue is usually quite thin but is must be divided in order to permit of an adequate approach to the saccus. In a left lobectomy this bar is divided bluntly along the dorso-medial margin of the left liver lobe in a resection of the middle lobe in which the left lobe is left behind it is divided near the margin of the quadrate lobe.

In cancer of the gall bladder the first parts of the liver to be affected are the adjoining liver lobes. This area shows numerous anastomoses between the blood and lymph vessel systems of both organs, which may be considered the pathways along which the spread of the tumour growth into the liver took place. As will be pointed out a resection of the pars dextra enables us to remove all diseased parts of the liver in early carcinoma of the gall bladder.

We developed a *middle hepato lobectomy* on a theoretical experimental basis an operation which seems to be suitable for resection *in toto* of the medial lobe of the liver together with the gall bladder.

As some of the principle of this operation have been developed in the previous chapters (p 170 and 182) we shall now only briefly consider the operative procedure itself.



Fig. 113. Right hilum of the liver after removal of the capsule in the right incision. 1. Ligature around the right portal vein. 2. Ligature around the artery and the bile duct which crosses the base of the right paramedian vein.

First of all the Glissonian pedicles of the cystic area of the right paramedian lobe and the crural area of the left paramedian lobe are exposed after which ligatures are applied around these structures but are not tied

The middle hepatic vein is dissected out at the crural pole of the liver and ligated which is followed by engorgement of the middle lobe, as soon as the area of the right interlobar fissure is clearly demarcated, the Glissonian pedicles are ligated The liver is opened bluntly along the right interlobar fissure which runs slightly to the right of the line of demarcation minor branches running from the right paramedian lobe to the right hepatic vein are ligated individually

The left interlobar fissure and the dorsal fissure are opened in the same way in a downward direction as far as the sagittal part of the left portal vein At this moment the Glissonian pedicle of the ventrocranial part of the left paramedian lobe is attended to It is dissected out at the ventral aspect of the sagittal part ligated and divided Thus the whole medial area is freed from the surrounding parts and can be taken out after Glisson's capsule and the containing structures have been dissected out of the transverse fossa

Care should be taken to avoid damage to the right and left hepatic veins and the hilar structures both during dissection and removal of the resected tissue

Still being in doubt as to the practical efficacy of a right hepato lobectomy, which, moreover seems to us a very tricky operation to perform we shall for the present leave it out of discussion

Resection of the pars dextra hepatis in which the left lobe and the caudate lobe are left behind can only be performed if sufficient liver tissue is left to support life The left lobe may occasionally be completely absent (Hammer 1928 Benz 1932)

In this operation the right hepatic artery right hepatic duct

and right portal vein are identified and ligated near the right hilar margin. In the umbilical fossa the capsule is incised along the quadrate lobe, the Glissonian pedicle of the caudal area of the left paramedian lobe is dissected out and widely ligated. The right and medial hepatic veins are dissected out and ligated at the cranial pole of the liver. The liver is opened along the left interlobar and dorsal fissure in a downward direction as far as the sagittal part of the left portal vein. At this moment the Glissonian pedicle of the ventro cranial part of the left paramedian lobe is attended to. It is dissected out at the ventral aspect of the sagittal part, ligated and divided.

The pars dextra is now rotated so that the hilum can be approached from the front and its structures dissected out of the transverse fossa. The Glissonian pedicle to the right liver half is ligated and divided after which the whole pars dextra is freed from the surrounding parts and can be taken out.

The raw liver surface is then re-peritonealized.

Chapter VI

THE CARE OF THE PATIENT BEFORE, DURING AND AFTER HEPATIC SURGERY

The fundamental principle upon which hepatic surgery was at one time thought to be based is the organ's tremendous regenerative capacity—an attribute which even the ancients probably recognised as witness the Greek myth of Prometheus. It will be remembered that as a punishment imposed upon him by the gods part of his liver was picked out every day by a bird of prey and according to the story was able to survive for centuries because the liver restored itself completely over night.

Since Gluck (1884) and Ponfick (1889) resected large parts of the liver in animals, it became common knowledge that up to 80% of the organ can be removed without ill effects to the organism while the remnant regenerates to such an extent that the organ regains its pre-operative weight within a relatively short period of time.

This regeneration however is subsequent to resection. Yet the patient lives on and as a rule the residue of the liver continues to function normally.

Indeed as Schalm and co-workers (1951–1954) have pointed out the fact that a patient is able to tolerate so extensive a resection is not due primarily to the regenerative power of the liver but to its functional reserve capacity which enables the organism to function normally on 20% (or more) of the normal liver tissue. They ascertained that when a large part

of the organ is functionally 'lost', the various function tests however subtle, rarely show any alterations, provided the liver was normal to start with.

Regeneration only takes place secondarily! Hence it is of the utmost importance that the organ should be functionally in the optimum condition when part of it is to be resected, because the 'reserve capacity' of the organ will decrease as its condition deteriorates. In other words, the function of a normal liver can be carried on by a remainder representing at least 20% of the normal weight of the organ. However, an extensive resection of this kind performed upon a liver in a worse condition may result in failing compensation of its functional capacity which is clinically manifested as 'hepatic failure'.

Schalm and co workers explain along these lines why the function tests made upon a liver affected by localised pathology—as contrasted with livers affected by generalised hepato cellular disease—are usually normal.

Acceptance of this conception implies, if surgery is decided upon, that it becomes a duty to make every effort to increase the functional reserve capacity of the organ so as to avoid the risk of a post operative hepatic failure, for which we hope to provide a number of rules in this chapter.

As has been stated, Ponfick was able to remove two thirds to four fifths of the liver, following which the animal recovered while developing hypertrophy of the spared part. New liver tissue was observed within about five days, and the average pre operative weight was recovered after six weeks or thereabouts (dogs). In patients this period was within 70 days (Locatello & Sitz 1950).

The regenerative capacity of the liver is reduced if the free drainage of bile and the portal blood supply are hampered. Nasse (1894) and later Mann *et al* (1920-29), and Schalm *et al*, demonstrated that blocking of bile ducts results in atrophy of the parts of the liver deprived of their ability to give off bile via these ducts. This atrophy however will be com-

compensated by the hypertrophy of the other parts of the liver left with free biliary outflow. Similar results were obtained after occlusion of one of the branches of the portal vein (Mann *et al*, Schramm *et al*).

Livers of young rats regenerate more rapidly than those in old rats (Buchner *et al* 1917).

A group of rats was put on a low protein diet for some time and then subjected to a 70% hepatectomy. This led to the death of most animals (Gurd & Vars 1949).

Mann *et al* who made a thorough study of the problem of regeneration, pointed out that restrictions of the restorative power of the liver are largely dependent upon a decreased portal blood flow and injuries to the liver such as cirrhosis restricting the portal blood flow will further aid in this characteristic.

The regenerative process is favoured by high carbohydrate diets (Davis & Whipple 1921) high protein diets (Gurd & Vars 1948 Vars & Gurd 1947) diets rich in vitamins (Denton & Ivy 1948), and by some hormones such as adrenal cortical extracts, thyroid hormone in moderate doses and testosterone. Occlusion of the arterial blood supply does not seem to exert any adverse influence on the regenerative processes in the rat (Wiles *et al* 1952).

It is not as imperative as in the case of the lungs where every type of resection is attended by a loss of function to choose the smallest possible hepatic territories for resection (*i.e.* segmentectomies), thanks to the great functional reserve capacity of the organ and its regenerative power. At present therefore we are not in favour of segmental resections for the following reasons:

- (1) The morbid conditions in the liver are only rarely limited to one segment, generally extending across the segmental borders into the adjacent segments.
- (2) Segmental resections are technically much more difficult to perform (and therefore are more hazardous to the

patient) than somewhat more extensive resections, which were discussed previously

A SOME REMARKS ON THE PREPARATION OF THE PATIENT BEFORE SURGERY

It is imperative to obtain some insight into the pre operative state of the organ. The first step is to enquire into the dietary history of the patient. It is highly important to record not only the quantity, but also the quality of the foodstuffs usually constituting the patient's diet. The daily consumption of meat (especially as regards fat meat and gravy), fish and fowl, fruit and vegetables, dairy products, condiments and spices (pepper, paprika, sambal, vinegar, curries, mustard, etc.) should be known. These data can be obtained from the patient or his family. Tactful questioning may reveal the patient's habits with respect to alcoholic beverages, a knowledge of kind and amount of the daily alcohol is also required.

Anorexia and loss of weight may be indicative of protein deficiency, this may be especially the case if there is a concomitant hypoproteinemia.

Our decision whether operation should be performed at once or postponed until corrective measures have taken their effect, is partly based on the knowledge gained from the patient or his family. Optimal nutritional conditions are imperative, as they constitute the most important prophylactic factor, helping in protecting the liver against damage by various noxious substances and in promoting the regeneration of hepatic tissue.

The liver is highly susceptible to dietary deficiency, frequently caused by dietary imbalance, the resulting changes in the liver cells may be indistinguishable from those caused by toxic agents or anoxia. These deficiency conditions should therefore be corrected by diets rich in carbohydrates, proteins and vitamins. A few hints may aptly be given here regarding the daily consumption of these foodstuffs.

Carbohydrates Glycogen besides exerting a protecting effect on the liver cells also may act as a protein sparing agent. On feeding 100 grams of protein, 38% of these proteins ingested are spared conversion to carbohydrates if the carbohydrate intake is adequate (Bollman 1931).

Woodlyth *et al* (1913) found that 60 g of intravenously administered glucose per hour could be utilized by a normal man without the development of glucosuria. This is 45% higher than the basic metabolic requirements which are mainly stored in the liver in the form of glycogen. Opie & Alford (1914) proved in animal experiments that though the liver is able to accumulate considerable amounts of glycogen in normal cases in the event of liver damage higher blood glucose concentrations are required before glycogen can be stored. In the latter case intravenous administration is necessary to supplement the oral intake. This method of administration is also preferred in case of vomiting or when early surgery is indicated and there is only a short time available for proper preparation.

The amounts of carbohydrates given daily should vary between 300 and 500 g (Morrison 1937). Foods rich in carbohydrates are potatoes, fruits, vegetables, sweetened fruit juices, cereals, honey, candy, jam etc. If the intravenous route is chosen for the reasons mentioned above, invert sugar (composed of equal parts of dextrose (D glucose) and laevulose (D fructose)) is better than dextrose.

Proteins Daily amounts of at least 100 g protein have been advised, some authors go even as far as recommending daily intakes exceeding 200 g (Morrison).

The importance of this dietary component in the conditioning of the liver against injury caused by noxious agents has been emphasized by various investigators (Radwin *et al* 1929, Himsworth & Glynn 1944, Smith *et al* 1928 etc.).

The process of detoxication is only partially understood; it may take place by conjugation of exogenous poisons and amino-acids by the absorption of toxic substances by plasma proteins (bile salts) through immunity or resistance to infections (globulin fractions) or by enzymatic processes in which proteins are required for the formation of the enzymes. Mobilization of fat from the liver cells decreases the susceptibility of these cells to injuries; certain amino-acids, the so called lipotropic agents acting as methyl donors in the process of transformation of fat into plasma phospholipids are indispensable (choline, methionine).

The liver is able to cope with excessive amounts of proteins (Dock 1947). Milk, cheese, lean meat, chickens and eggs are foods rich in proteins; milk in particular is to be recommended (Schalm 1951).

In view of the fact that one litre of whole blood contains only about 150 grams of protein, it will be evident that the amount which can be administered in this way is insufficient to correct a severe state of hypoproteinæmia.

Oral intake or tube feedings are therefore imperative, but additional intravenous administration of whole blood is indicated in cases of anaemia and hypoproteinaemia because these patients are more susceptible to

post operative haemorrhagic or traumatic shock (Ravdin *et al* 1951)

The most important factor to be borne in mind in the composition of a diet is its palatability. It is therefore impossible to eliminate all fats (Hoagland 1945). Small amounts of fat not only improve the savoriness of the diet but moreover promote the utilization of carbohydrates as well as of proteins (Elvehjem *et al* 1947).

Feeding by stomach tube is occasionally necessary for those patients who are unable to eat or who are unwilling to take up sufficient quantities. The food mixtures used should always be prepared freshly in order to prevent contamination.

Vitamins are as essential in the diet as proteins—both are needed for the production of enzymes.

The daily thiamin requirement—necessary for lactic acid metabolism—is 10–20 mg by mouth or intravenously and of niacinamide 50–100 mg. Nicotinamide deficiency may lead to hepatic insufficiency with concomitant porphyrinuria (Rafsky & Newman 1943). Folic acid seems to possess a lipotropic effect. Vitamin C promotes the utilization and storage of carbohydrates in the liver and participates in the intermediary metabolism of some amino acids (tyrosine). It decreases the deposition of fat in the liver and protects it against damage. Liver extract is a powerful protective substance and promotes the regeneration of this organ (Forbes 1939).

The relationship between prothrombin concentration of the blood and vitamin K is well known.

A number of medical conditions influence the operative risk *e.g.*, endocrine disturbances such as diabetes, hyperthyroidism, moreover cardiac, hepatic and renal affections.

Generally speaking, evaluation of the patient and his cardiac, hepatic and renal reserves, and correction of the functional disturbances of these organs as far as possible, are necessary if the body is to be able to cope with the strain imposed by hepatic surgery. Examination of the patient by a specialist of internal diseases is therefore a wise policy.¹

B DANGERS AND SAFEGUARDS DURING SURGERY

During surgery a number of problems arise such as prevention of air embolism, the choice of the anaesthetic, the reaction of the organ to manipulation at one of its gateways etc. which will be discussed here briefly.

In 1911 Fiegel instituted an investigation into air embolism

after observation of a case in which this complication arose due to the accidental opening of a large vein in the intracavitary area. The negative intrathoracic pressure proved to be transmitted to the venous system in the immediate neighbourhood of the chest as appeared from his experiments. Upon opening the thoracic cavity the negative pressure falls immediately which reduces the chance of air embolism. On intubating the patient controlled respiration can be applied. The latter can easily be used for re-breathing under positive pressure which in Tiegel's experiments, led to a rise of the intracavitary pressure. In this way air embolism can be prevented.

To determine the pressure in the hepatic veins pressure readings were taken in these veins with closed thoracic cavity during cardiac catheterization (De Vos 1934). The average pressures varied between zero and $+1$ cm H_2O being negative during inspiration and positive during expiration.

During a hemi-hepatectomy performed with closed chest opening of the hepatic veins resulted in slight bleeding during expiration which stopped completely during inspiration. A withdrawal of blood was actually observed during the latter phase. The negative intrathoracic pressure during inspiration is transmitted to the hepatic vein system. Prevention of air embolism by re-breathing under positive pressure has the disadvantage of increased venous bleeding due to a rise in venous pressure. We therefore no longer sever the hepatic venules before they are ligated. This can only be done by dividing the liver bluntly which we first attempted in dogs with satisfactory results. Furthermore it is advisable to intubate patients who are undergoing liver operations.

Anaesthesia produces a considerable hypoglycaemia (Lannhauser 1916) there is evidence that this is due to hepatic glycogenolysis (Mekie 1931). The parenteral administration of glucose not only during the pre and post operative periods but also during the operation has been emphasized by Pereira

& Probst (1949), who studied the degree of assimilation of infused glucose. The maximum hyperglycaemia occurs immediately after the operation, followed by a fall towards the pre-operative blood sugar level, which is usually reached within about eight hours. There is a close relationship between the absolute rise of the blood sugar level and the duration of anaesthesia. In cases of post-operative shock the blood sugar level is also high.

Moreover, the liver plays an important part in the inactivation or detoxification of numerous substances. It is therefore not surprising that the duration of the activity of thiopental (pentothal) is considerably prolonged in patients with liver affections (Shideman *et al.*, 1949). Cats, partially hepatectomized after anaesthesia with certain drugs (chloral hydrate, brometol, thiopentone and soluble phenobarbitone), remained unconscious for a longer time than normal animals (Walker & Wynn Parry, 1949), which shows that these substances are detoxified to some extent in the liver. This fact ought to be taken into account while planning an extensive resection.

Some anaesthetics exert an adverse influence on the function of the liver. Bourne *et al.* (1930), for example, observed impaired liver function for a period of eight days, caused by 30 minutes chloroform anaesthesia. Two hours anaesthesia with this drug led to alterations in the hepatic function for six weeks. Chloroform is therefore the most notorious hepatotoxic anaesthetic agent.

Ether anaesthesia causes only a slight depression of the liver function, 15% dye retention is found after 24 hours in the B.S.P. test, but the values returned to normal after 48 hours.

Bourne (1939) demonstrated that nitrous oxide or ethylene anaesthesia of 1-2 hours duration did not cause immediate or remote impairment of the hepatic function if sufficient oxygen was administered at the same time, of all animals the function was affected. However, if the percentage of oxygen administered was deliberately reduced the values did not return to

normal in a short time as in other anaesthetics about six days being required for recovery

Cyclopropane has absolutely no adverse influence on the liver function, as shown by Raginsky & Bourne and is therefore the anaesthetic *par excellence* being not only perfectly suitable for patients with hepato cellular diseases but also in cases of hepatic surgery

The condition of the liver is the determinative factor for the response to the anaesthetic agents the adequacy or inadequacy of the patient's diet before the anaesthesia is another important factor in this respect

The prevention of anaemia during surgery is a pre requisite (Mekie 1931)

The trauma caused by the manipulations during surgery results in a depression of the hepatic function Lord (1939) noticed a fall in the plasma prothrombin concentration after surgical exploration of the liver

A negative nitrogen balance is usually observed after any major operation A significant fall of the serum albumin level may also occur frequently persisting for some time after the operation it should be corrected by oral or tube feeding with additional transfusion of whole blood human plasma or if necessary human serum albumin General and local trauma together cause a stress situation leading to impairment of the liver function the degree of the damage depends on the pre operative condition of the organ and the strain imposed on the individual Depletion of the carbohydrate and protein reserves makes the liver extremely susceptible

Manipulation on retraction or compression of the vascular pedicle entering the liver may result in massive or confluent centrilobular necrosis due to impediment of the hepatic blood flow and anoxia of the liver in addition to the strains mentioned before

Burton Opitz (1911) found the following figures in animal experiments: about 40% of the oxygen required is transported by the hepatic arterial blood, which constitutes only about 25% of the total amount of blood reaching the liver. The portal vein carries about 75% of the total amount of blood, contributing with 60% to the total oxygen saturation.

The previous paragraph shows that the *hepatic artery*, although carrying only a minor part of the total hepatic blood supply, is of very great importance as regards the oxygenation of the liver. A great many publications on the results of occlusion of this artery have been published since Malpighi's observations in 1699, who noted in animals that ligation did not interfere with the secretion of bile.

As pointed out by Von Haberer (1905), proximal ligation of the vessel is readily compensated by numerous collaterals.

We have already discussed that, under normal conditions most of the branches of the hepatic artery at the hilum are end arteries in the sense of Cohnheim. Interruption of the proper hepatic artery distal to anastomoses with other vessels (gastro-duodenal artery, right gastric artery, etc.) or at the hilum of one of its branches may lead to changes varying from very mild ones to extensive necrosis of the areas deprived of their arterial blood supply; the latter condition is usually associated with necrotic changes in the wall of the gall bladder.

Dogs which died after ligation of the hepatic artery, if the site of ligation was distal to the anastomoses mentioned (Von Haberer 1905) Narath (1915) was able to prevent death by anastomosing the hepatic artery with the portal vein. Liver protecting agents were entirely unable to change the course of events (Barritt *et al.* 1952), which means that the acute hepatic necrosis, with or without rupture of the gall bladder that followed the ligation was apparently of a toxic rather than of a simple nutritional nature.

In 1909 Wolbach & Suda demonstrated the presence of spore bearing anaerobic micro organisms in healthy dogs

this finding was confirmed by Markowitz in 1949 and by many others subsequently. This fact explains the septic clinical manifestations under which these dogs when deprived of their arterial blood supply, died within a period varying from 12-60 hours.

The picture was completely changed however by the use of antibiotics especially penicillin and aureomycin (Markowitz). Untreated dogs show a fatal septic necrosis of both liver and gall bladder, the portal vein branches being filled with masses of bacteria (Hartroft 1947). Of the dogs treated with antibiotics about two thirds of the survivors showed a diminished hepatic function during the first few days, but the hepatic insufficiency had disappeared by the 12th day after operation (Irizer *et al.* 1951). This leads to the assumption that the antibiotics prevent the aerobic micro organisms normally present in the dog's liver from growing and releasing their toxic products on the susceptible liver cells during the period of hypoxia between the moment of hepatic artery ligation (Markowitz 1949) and the time when the collateral circulation is probably able to take over (Von Harberer 1905 Segall 1923).

Though Romieu & Brunschwig (1951) were unable to demonstrate similar micro organisms in the liver of healthy human beings, Lowe *et al.* (1951) found that several livers of patients who died after hepatic arterial ligation contained gas probably originating from a fulminating infection by anaerobic gas producing bacilli which are normally present in the intestines and probably transported to the liver via the portal vein or biliary canaliculi.

Though formerly compression of both hepatic artery and portal vein was recommended during liver surgery (Baron 1910) at present it is realized that this can only be done as a last resort—and then for as short a time as possible.

Sudden occlusion of the *portal vein* causes a transient shrinkage

of the liver, with return to normal within a few minutes without any change in colour. Compression with total occlusion of the portal vein and its results are discussed by Peck & Grover (1952). These authors observed a sharp fall of the arterial blood pressure immediately after sudden occlusion of the portal vein. This fall appeared to be of a reflex nature and it was followed by a further but gradual reduction of the blood pressure, which caused death in deep shock within an hour. This secondary fall was apparently due to changes in the blood volume. The impression was gained from these experiments that the initial fall of the arterial blood pressure originated in the splanchnic mesentery and not in the liver. A temporary suppression of this response could be effected by means of corticotrophin (ACTH), but not with cortisone. The gradual secondary decrease is a result of blood pooling within the peripheral portal system.

Gradual occlusion does not elicit this response, if a sufficiently long period is allowed for the development of collaterals.

However, in a number of cases, recently operated on (pancreatico-duodenectomy) by Child *et al* (1950), deliberate sudden occlusion of the portal vein by means of ligation was tolerated in both monkey and man. The blood pressure in most instances returned to the pre-operative values within 20-30 minutes.

We have seen that the temporary or permanent occlusion of the portal vein and proper hepatic artery often cannot be tolerated and it remains to add that in many cases the occlusion of branches of the veno-hepatic system may lead to fatal results. This depends largely on the area of the liver that is deprived of its means of blood drainage.

Obstruction of the hepatic veins (Budd-Chiari Syndrome), though in general due to a primary endophlebitis from the hepatic veins or to obstruction by tumour or abscess, may be the result of a technical error during surgery. Occlusion of one

or more branches may result in the acute form of the syndrome. It is characterized by a sudden access of abdominal pain, nausea and vomiting. The liver and spleen enlarge rapidly, massive ascites appears, usually followed by death in hepatic coma within one to four weeks. The diagnosis ante mortem is difficult: only ten of the cases reviewed by Thompson (1947) were diagnosed ante mortem. Gradual occlusion of the various trunks of the hepatic vein is accompanied by atrophy of the part which is unable to drain its blood and followed by hypertrophy of the residual normal organ (Murray & Himmel 1949). From these facts it is clear that during surgery care should be exercised to avoid injury of the major hepatic veins marking the plane of resection.

Although occlusion of smaller vessels may often not have fatal results, the need for careful preparation at both gateways of the liver will be obvious from the preceding paragraphs.

Upon completion of the operation we advise leaving one drain in the subdiaphragmatic area and another against the liver in the region of resection. The removal of these drains is effected in the same way as in other fields of surgery.

C ON SOME COMPLICATIONS IN THE POST OPERATIVE PERIOD

Many complications may occur in the post operative period, namely those associated with this special type of surgery in particular and those incident upon any type of surgery, generally. We shall be mainly concerned with the former, the latter can be found in any textbook on this topic. Our subjects will be post operative shock, portal vein obstruction, cholerrhagia, pulmonary complications, paralytic ileus, liver cell embolism, infection and hepatic failure.

Shock has a very adverse effect on the condition of the liver.

Friedman *et al.* (1951) observed radiologically a narrowed intrahepatic portal vascular bed in the dog while Seneviratne

of the liver, with return to normal within a few minutes without any change in colour. Compression with total occlusion of the portal vein and its results are discussed by Peck & Grover (1952). These authors observed a sharp fall of the arterial blood pressure immediately after sudden occlusion of the portal vein. This fall appeared to be of a reflex nature and it was followed by a further but gradual reduction of the blood pressure, which caused death in deep shock within an hour. This secondary fall was apparently due to changes in the blood volume. The impression was gained from these experiments that the initial fall of the arterial blood pressure originated in the splanchnic mesentery and not in the liver. A temporary suppression of this response could be effected by means of corticotrophin (ACTH), but not with cortisone. The gradual secondary decrease is a result of blood pooling within the peripheral portal system.

Gradual occlusion does not elicit this response, if a sufficiently long period is allowed for the development of collaterals.

However, in a number of cases, recently operated on (pancreatico duodenectomy) by Child *et al* (1950), deliberate sudden occlusion of the portal vein by means of ligation was tolerated in both monkey and man. The blood pressure in most instances returned to the pre-operative values within 20-30 minutes.

We have seen that the temporary or permanent occlusion of the portal vein and proper hepatic artery often cannot be tolerated and it remains to add that in many cases the occlusion of branches of the veno-hepatic system may lead to fatal results. This depends largely on the area of the liver that is deprived of its means of blood drainage.

Obstruction of the *hepatic veins* (Budd-Chiari Syndrome), though in general due to a primary endophlebitis from the hepatic veins or to obstruction by tumour or abscess may be the result of a technical error during surgery. Occlusion of one

or more branches may result in the acute form of the syndrome. It is characterized by a sudden access of abdominal pain, nausea and vomiting. The liver and spleen enlarge rapidly massive ascites appears usually followed by death in hepatic coma within one to four weeks. The diagnosis ante mortem is difficult only ten of the cases reviewed by Thompson (1947) were diagnosed ante mortem. Gradual occlusion of the various trunks of the hepatic vein is accompanied by atrophy of the part which is unable to drain its blood and followed by hypertrophy of the residual normal organ (Murray & Kimmel 1949). From these facts it is clear that during surgery care should be exercised to avoid injury of the major hepatic veins marking the plane of resection. Although occlusion of smaller vessels may often not have fatal results the need for careful preparation at both gateways of the liver will be obvious from the preceding paragraphs.

Upon completion of the operation we advise leaving one drain in the subdiaphragmatic area and another against the liver in the region of resection. The removal of these drains is effected in the same way as in other fields of surgery.

(c) ON SOME COMPLICATIONS IN THE POST OPERATIVE PERIOD

Many complications may occur in the post operative period namely those associated with this special type of surgery in particular and those incident upon any type of surgery generally. We shall be mainly concerned with the former the latter can be found in any textbook on this topic. Our subjects will be post operative shock, portal vein obstruction, cholelithiasis, pulmonary complications, paralytic ileus, liver cell embolism, infection and hepatic failure.

Shock has a very adverse effect on the condition of the liver. Friedman *et al* (1951) observed radiologically a narrowed intrahepatic portal vascular bed in the dog while Seneviratne

(1949) found a narrowing of the sinusoids during hypotensive conditions following haemorrhage in the frog

Wiggers (1946) found a reduction of the portal blood flow through the liver during periods of hypotension

It is assumed that hepatic anoxia is followed by a narrowing of the sinusoids or else by swelling of the liver cells to such an extent that the hepatic cells are no longer accessible to adequate amounts of oxygen. In course of time a vicious circle develops and the changes may become irreversible (irreversible shock)

If the condition of shock lasts longer than 24 hours, the initial swelling of the liver cells results in a centrilobular liver cell necrosis (Ellenberger *et al*, 1951)

In Delorme's experiments (1951), in which the liver of dogs was perfused with oxygenated arterial blood while the animals were kept at a given level of hypotension, these dogs showed a marked increase in resistance to the duration of hypotension as compared with controls, up to prolonged periods

Narrowing of the intrahepatic portal vascular bed may moreover occur after a variety of conditions in which the liver cells are damaged, such as inadequate diet, incorrect anaesthesia, surgical trauma, infection, loss of blood, respiratory troubles, ileus, etc., all these conditions are commonly encountered after surgery

It is easy to see that these various factors may well tend to render a post operative shock caused by trauma, haemorrhage etc. irreversible. When treating for shock it is necessary, in addition to coping with the direct cause of the shock, to deal vigorously with any damage the liver may have suffered, using whole blood and oxygen. It has been found, moreover, that antibiotics like penicillin and aureomycin are useful in the treatment of shock (Fine *et al*). It is assumed that micro-organisms bearing micro organisms have an opportunity of proliferating during the period of hepatic hypoxia and this can be prevented by administering antibiotics

Portal vein thrombosis Of the three main types, described by De Josselin de Jong (1912) both the truncular and the terminal type may occur, the former affecting the main stem of the portal vein the latter the intrahepatic branches. The number of cases reported in patients operated on by the new method of hepatic surgery is very small, but both Fortot Jacob *et al* (1952) and we instituted anti coagulant therapy. Nothing definite can therefore be said of the incidence or the initial symptoms of this complication of hepatic operations. Nonetheless it should be kept in mind and one should not hesitate to start anti coagulant treatment if any of the three symptoms *abdominal distension splenomegaly and ascites* should arise.

Paralytic ileus Distension of the intestine especially of the stomach causes impairment of the liver function in rats due to the impeded hepatic blood flow this was shown by Estrada *et al* (Le Riden & Vars). Intestinal obstruction per se has been the cause of simultaneous failure of liver and kidney function resulting in some instances in acholia and anuria (hepatorenal syndrome Schnedorf & Orr 1942). It is not known whether this is due only to abdominal distension. Following surgery on the liver therefore continuous Wainman suction is indicated in all cases.

Pulmonary complications It is imperative to maintain an open airway and to prevent pulmonary complications such as atelectasis of the right lower or middle lobe or bronchopneumonia which might give rise to a condition of hypoxaemia. Bandages should not impede easy breathing and affect the adequate respiratory exchange. The importance of deep breathing and coughing must be explained to the nursing staff in order that they encourage the patients to do so. The semi sitting posture which allows more free breathing to the patient and relieves the tension of the wound should be alternated with the supine position and turning the patient on

the opposite side every two to three hours. Any drugs exerting a harmful effect on the respiration should be avoided. Regular chest examinations, especially on the right side and in case of doubt, X ray examination, are necessary.

The occurrence of *liver cell embolism* following hepatic trauma is a complication unfamiliar to most authors. Jurgens (1886), Von Rieckhnghausen (1886) and Schmorl (1888, two cases) were the first to recognize the occurrence of embolic disseminations of liver fragments following traumatic injury to this organ. According to Kaufman, the presence of liver cell embolisms in the pulmonary artery is not an everyday observation and has so far been observed only microscopically (1931). This is not altogether correct, because the first case described by Schmorl was detected macroscopically in the pulmonary artery. The same observation was made by Willer (1935). Usually, however, the emboli are only detectable microscopically, but they may still be the cause of death in the event of massive dissemination via the open hepatic veins. It mostly occurs after crushing external injuries, but also following hepatic surgery. The incidence of pulmonary embolism after liver surgery is high, and several of the cases reported were probably due to this cause.

We therefore once more wish to emphasize the importance of blunt division of the liver during operations, with ligation of the hepatic venules crossing the plane of resection, before their division, a technique which we imagine, will at least partly circumvent this complication (see p. 174).

(Cholerrhagia) Leakage of bile with localized abscess formation, generalized biliary peritonitis, biliary empyema or biliary fistulae have been described, especially in the period when conservative treatment of lacerations was still the rule. Normal bile does not seem to cause as much harm as infected bile, the first is either encapsulated and slowly resorbed, or it gives rise

to an aseptic chronic peritonitis. Infected bile causes fulminating peritoneal sepsis, leading to rapid death (Noetzel, 1910). All free bile should be removed during the operation, and there should be a careful check on leakage by compressing the gall bladder (see p. 180).

Infections. The frequency of abscess formation is reduced by leaving drains in the subdiaphragmatic space and against the area of liver resection. In this type of surgery, however, some devitalized liver tissue will always be spared, because by carrying the resection too far, healthy tissue will be involved in the end, resulting in haemorrhage.

Although we do not wish to advocate the routine use of antibiotics, we still feel that these therapeutic agents (usually penicillin or aureomycin) should be given for a period of 7-10 days after the liver operation. This opinion is based on the observations of Markowitz and others mentioned before.

The devitalized liver tissue may have a twofold effect. The first as a toxin released from the tissues by autolysis affecting both liver and kidneys (Mason *et al.*) and the second on an inflammatory basis due to fulminating infection of dead tissue (Markowitz). Whether pre-operative sterilization of the intestines which is most probably the source of infection ought to be recommended is an open question.

Every abdominal infection as shown by Serege, Wassink, etc., is a severe drawback upon the liver and ought to be treated vigorously.

Reverting to our point of departure it appears that the normal liver is endowed with a superlative reserve capacity which may however be impaired by a variety of factors. As we have seen, some conditions may recoil upon the general condition of the organ and reduce its reserve capacity.

Any functional insufficiency there may be in the residual part of a resected liver is seldom due to one specific factor.

Usually it is a number of coincident factors which are jointly responsible for the injury suffered by the cells of the liver.

Disturbed hepatic function tests have shown that the functions of the liver are subject to failing compensation. It is incumbent upon us to bring all our knowledge and energy to bear on the prevention of this *hepatic failure* by eliminating, as far as we can, every incipient cause of such a condition. Hence the necessity for proper pre- and post-operative treatment of a patient undergoing hepatic surgery.

Heyd, Boyce & McFetridge, Helwig & Orr, who made extensive studies of hepatic insufficiency after operations distinguish two forms of hepatic failure. The first form, the acute type (liver death, Heyd 1923¹), usually becomes manifest immediately after the operation, it is characterized by hyperpyrexia, and tachycardia, associated with rapid development of lethargy, stupor and coma, death following within 18-36 hours after the operation. Marked degenerative hepatic changes are generally found in this type.

The second form is usually a subacute or chronic type of hepatic failure, mostly arising several days to weeks post-operatively, if the functional decompensation resulting from surgical trauma is not counteracted. Death follows within 10-14 days or even longer. Symptoms of severe renal insufficiency predominate in addition to more or less pronounced evidence of hepatic insufficiency: progressive oliguria and the appearance of albumin casts and frequently erythrocytes in the urine. Helwig and Schutz describe the clinical course as follows:

The patient usually lapsed into a muttering delirium which rapidly progressed into coma and the nitrogenous elements of the blood greatly increased, while the urinary nitrogen strikingly decreased. At about this time nausea and vomiting sometimes became prominent symptoms. These latter symptoms were most marked in the post-operative cases from the 5th to 8th day when with the removal of the stitches

a decided delay or even absence of wound healing was often observed. Practically always some mucous surface bleeding was noted and in many instances it was so striking that the vomitus and stools consisted almost entirely of blood. The clinical picture then progressed, as a rule to that of profound uraemia. low grade generalized oedema developed and almost total anuria followed. The retention of the nitrogenous products in the blood became more marked and the patient died in a state clinically resembling uraemia.

This description is exactly that of the chronic hepatic insufficiency syndrome.

Similar to cardiac decompensation which occurs when a new burden is thrown upon the circulation the liver is able to maintain a standard pattern provided that the reserve capacity is not needed. however when the patient is subjected to a surgical operation the liver has no reserve capacity for sustaining this new burden. Thus the liver permits the normal toxic substances from the intestinal tract to pass undetoxified into the general circulation. These impurities plus toxic metabolites from necrotic liver cells overburden the kidneys with attempt to take over the duties of the liver (Boyce *et al*). When the kidneys are thus forced to assume the detoxifying function of the liver this added physiological necessity results in renal decompensation. Hence it can be appreciated that initial hepatic exhaustion produces renal insufficiency and death (Ficarra 1950).

Both syndromes are the manifestations of the same process.

We believe that the so called liver death or hepatorenal syndrome is a single pathological process in which the hepatic changes always precede the kidney changes. In our opinion if the patients who die promptly with hyperpyrexia and who exhibit liver degeneration at autopsy could be kept alive long enough they always would show precisely the same clinical and post mortem renal changes as do the patients who die later with typical symptoms of uraemia. (Boyce *et al*).

Changes in the liver and the kidneys (convoluted tubules) vary from a light to severe degeneration

Prophylaxis should aim at preventing a large residue of devitalized liver tissue, because there is strong evidence that proteins, released from these devitalized parts, act as toxins, affecting both liver and kidneys (see p. 148)

We do not propose to deal *in extenso* with the treatment of the various complications; reference may be made to textbooks of internal medicine for treatment of the different forms of liver failure. Our aim has been to point out the risks to which the liver—and hence the patient—is exposed and, wherever possible, to suggest in brief how such risks might be averted.

Appendix I

EMPLOYED INJECTION CORROSION TECHNIQUE

The injection of intra organic hollow structures for the purpose of anatomical study was introduced around 1650 by some prominent Dutch anatomists, at a later date their methods were amplified by a number of corrosion techniques.

Joh van Horne professor of anatomy at Leyden University is becomes apparent (according to Hyrtl) from his *Novum ductus chyloferus nunc primum delineatus* Leidae 1652, in which he introduced this technique to demonstrate the thoracic duct which had been discovered some years previously by Jean Pecquet independently of Olaus Rudbeck.

Reynier de Grief (1673 Leyden) developed an anatomical syringe which he only used for some physiological experiments performed in an endeavour to prove the correctness of Harvey's theory of the circulation of the blood.

The brilliant Johannes Zwammerdam doctor of Leyden famous for *Algemeene Verhandeling van de Bloedelooze Diertjes* (Utrecht 1669) and *Miraculum naturae s. uteri muliebri fabrica* (Leyden 1672) not only described his method partly *ut uteri alique corporis cavitates (venae scilicet arteriaeque) ita praeparari possint ut genuinam suam faciem exhibeant et archetypi loco inscribant designandis earundem figuris* but also the material used for his injections *Resipe cerae albae quantum videbitur eamque liquefactum rubro flavo viridi vel alio colore tinge et siphone qui*

cochlear adstrictum tubulum habeat etc.' He was the first to describe the intrahepatic ramifications of bile ducts and arteries.

F. Ruysch corroded his injected specimens, but both methods are unknown, because he kept them secret.

Lieberkuhn (Berlin, 1750) described, next to a number of materials he used for injection purposes (mixtures of wax, with clophonium and turpentine added etc.), some corrosion media, such as esprit de nitre, assez fort (nearly concentrated nitric acid) or sulphuric acid, detempte dans l'eau, jusqu'à ce que l'acide ait dissous ce qui n'est pas dans la circulation, etc.

One of the greatest authorities on injection corrosion anatomy was Hyrtl, who made an extensive study of practically every hollow structure of the human body and of animals including the heart and its vessels, lungs, liver, spleen, kidneys etc. His book 'Die Corrosionsanatomie und ihre Ergebnisse' Vienna 1873 embodies the results of 12 years of injection corrosion anatomy.

The introduction of X rays in medicine enabled a method of investigation to be evolved (developed by Von Hildebrand 1907) by which radio opaque media are injected into the intraorganic hollow structures. Martens (1920) used this technique to study the intrahepatic course of the hepatic arteries. Segall (1923) did the same for all the intrahepatic structures following which this method was used by several other authors for the same purpose.

Since the discovery of plastics and similar products these materials have been used as an injection medium in the injection corrosion technique, at first applied to the study of the anatomy of pulmonary structures and the vessels of the heart more recently to that of the liver as well.

Method

A pressure chamber obtained from an old fashioned discarded autoclave was divided into four compartments (fig. 114). Each

compartment was drained by a metal tube. Pressure was applied by means of compressed carbon dioxide, which was passed through two pressure reducing valves, the second one



Fig. 114. Injection apparatus: 1 inlet valve; 2 four compartments; 3 four draining tubes with cocks.

being used to check the pressure in the chamber. After passing these valves, the gas proceeded via rubber tubings to an inlet at the top of the apparatus. The four compartments continued

plastic of different colours. The various hollow structures were cannulated by means of glass tubes and connected with the pressure chamber through rubber tubings. The livers, obtained at autopsy from the Department of Pathology of St. Cransius Hospital of Nijmegen, were thoroughly cleaned by mechanical means prior to injection. Moreover, the cancerous livers were insufflated with compressed carbon dioxide. During the injection, the liver floated on water. The portal vein and bile ducts were injected under a pressure of 0.1 atm, the proper hepatic artery with a pressure of 0.4-0.5 atm and the hepatic veins with a pressure lower than 0.05 atm. The arteries were first injected till the arterial vascular bed was completely filled, which was observed as a cessation of the flow of plastic in the glass connection pieces between the rubber tubings. Re injections followed in the next half hour, to compensate the shrinkage of the material. Subsequently, the bile ducts and portal veins were injected. The hepatic veins were the last to be dealt with.

The plastic was re injected several times on the same day and the following day. After complete solidification of the plastic, the liver was placed in a concentrated hydrochloric acid solution (45%) for 3-5 days. Removal of the remnants of liver tissue was effected by means of a fine jet of water from a nozzle. The plastic which we used was prepared in our laboratory from polyvinylite (supplied by Waterhouse Mason & Co. Rotterdam, Holland) and acetone.

2000 grams of polyvinylite were added to 5000 cc of acetone. The suspension was shaken by hand or with the aid of an adapted machine (as used in serology) until the solid was dissolved. We also used plastoid for injection purposes (supplied by Rohm & Haas Darmstadt, Germany). Pigments were added which met the specifications drawn up by Liebow *et al.* namely, they mixed well with the plastic, they were not affected by the concentrated hydrochloric acid and they did not bleed.

Those colouring materials were
red (hepatic artery) mercurio sulphide
blue (portal vein) prussian blue
yellow (bile duct) picric acid
white (hepatic vein) kaolin

Later we used the various pigments supplied by Rohm & Haas because they suited our purpose so much better

Material

Our study is based on over 100 coloured polyvinylite corrosion specimens of normal human livers 15 specimens of cerous human livers 3 dog livers and 1 pig's liver. The anatomy of the dog's liver will be discussed briefly and compared with the human liver, because part of our hepatic surgical experience was gained on dogs. In spite of the considerable differences in the intrahepatic structures of these two livers there is sufficient resemblance so that with these differences and similarities in mind the liver of the dog may be of great practical use for experimental purposes. Only a few of the organs were injected *in situ* mainly in order to study the intra and extrahepatic courses of the portal vein in their natural relationship to each other. Dissections of the hilum of approximately 25 fresh as well as injected specimens were carried out to study the hilar anatomy for surgical purposes. The nomenclature proposed in this study is restricted to the ramifications of the first and second order. It is a topographic al one comparable with those used in the past for other organs.

Appendix II

HEPATOGRAPHY

The practical implications of the anatomical data are certainly not only of value to surgery. In future they will undoubtedly be useful for diagnostic purposes as well.

Liver function tests, which are useful in the determination of the extent of liver damage and its response to treatment, usually fail to give any definite information in case of tumours of the liver (Thomas & Zimmerman, 1952).

Up to the present hardly any attempts have been made to study the anatomical peculiarities (see p. 170) or certain diseases (see p. 133) of the liver by injecting radio opaque substances into the hollow structures during operation and taking X ray photos of these structures. Yet operative cholangiography is used for the diagnosis of stenosis of extra hepatic ducts. may very well suit these purposes.

Several attempts to visualize the hepatic structures *in vivo* for diagnostic purposes have been described recently. These procedures may moreover be instructive as to the intrahepatic anatomy of one particular liver for instance one selected for operation.

Operation is avoided in *transabdominal cholangiography*, is recently developed by Carter & Szypl (1952). A needle or trocar is pushed slowly through the abdominal wall into the liver substance. If the needle pierces the wall of the duct, the resistance increases diminishing again if the point enters the

lumen of the duct. Withdrawal of the stylette at this moment is followed by flow of bile (especially in cases of obstructive jaundice). This method was initially tried out during exploration. The bleeding from the needle puncture and the drainage of bile from a dry tap were slight and subsided promptly.

Radio opaque substances, injected through the needle, will cause filling of all intrahepatic bile ducts, provided both hepatic ducts are open. However, transabdominal cholangiography performed along these lines seems to us a difficult and hazardous procedure.

Operative portal phlebography, and more recently *transabdominal portal phlebography* (Albertucci & Crump 1951) can be utilized for the same purposes though up to now it has only served for diagnosis of portal vein obstructions. Several reports have already appeared on its diagnostic use since its introduction (Jeger & Middlemiss 1954, Aurig 1954 and others).

The spleen is punctured with the patient under general anaesthesia, on the side of the anterior axillary line in the 9th-10th left intercostal space and a needle about 15 cm long is inserted in the antero-posterior direction, the patient holding his breath for a few seconds. If however some disturbance of the blood clotting mechanism is expected, the puncture is carried out immediately before the operation with a thin needle. A scratching sound is heard at the moment when the needle strikes the capsule of the spleen. Blood can be aspirated when this capsule has been passed. The back flow may be forceful in the event of portal hypertension. If dye is injected into the spleen this organ will contract under the influence of the stimulation and the dye will be distributed over the portal vein system. This method has only been used up to the present day for the diagnosis of portal obstruction and the examination of the collateral circulation. The procedure was carried out in dogs by Tori & Scott who did not find any macroscopical or microscopic histopathological changes of the spleen.



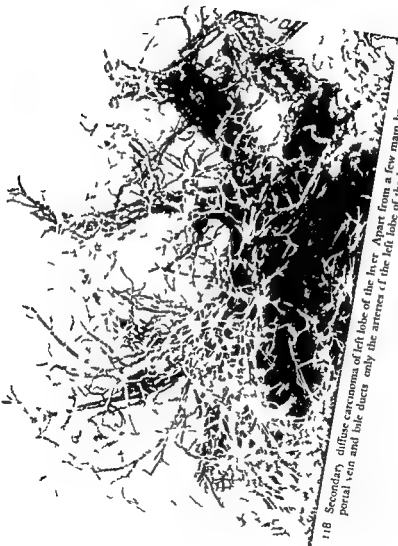
Fig. 11. Large lobular metastatic carcinoma of the liver (primary tumor in the breast). No filling of veins and arteries at this site.



Fig 116 Detail of the notch caused by the tumour in the previous photograph



Fig 11. Metastatic disease carcinoma. Only the arteries have filled in the area between the artery and the primary tumour carcinoma of the stomach.



118 Secondary diffuse carcinoma of left lobe of the liver. Apart from a few main branches of the portal vein and bile ducts only the arteries of the left lobe of the liver have filled



Fig. 110. Vessels in hepatic vein specimen at the site of a nodular carcinomatous metastasis.



Fig. 120 N du lar metastatic carcinoma of the liver (primary carcinoma of the rectum) Abn renal arteries in the growth Venu and bile ducts not filled in situ

Middlemiss, after using this method in 26 patients, did not observe any bleeding and the experience of all other investigators employing this procedure (Leger, Aurig, and others) was the same. If there is no extrahepatic portal obstruction, the major intrahepatic portal vein branches are filled in this way.

In 1952 Tori developed a method of visualizing the hepatic veins by passing a cardiac catheter into the inferior vena cava via the right auricle. By inserting the tip of the catheter alternately into the right and left hepatic vein, each vein was injected with 70% diodrast, the patient holding his breath as in the Valsalva test (to obviate backflow of the dye). Hepatic venograms produced in this way showed local pathology.

What is to be expected of the hepatogram of a patient with metastases in the liver and which structure, after injection, is likely to provide the most revelatory data for a diagnosis? In an endeavour to find the right answers to these questions, we also injected carcinomatous livers at the very outset of our research because this procedure enabled us to study the circulation of the blood in the affected part.

Breedis and Young (1954), who also investigated the blood supply of hepatic tumours, arrived at the conclusion 'that malignant neoplasms growing in the liver tend to acquire an exclusive arterial blood supply'. This opinion was based on the study of specimens of livers injected with India ink and coloured gelatin. Prior to the appearance of their paper, our results of the study of the injection corrosion specimens of livers—with secondary tumour growths varying from 1–10 cm in diameter in 15 specimens—were that there are two types of secondary tumour growths, each of which shows a different relationship to the hepatic artery.

The first type is that usually encountered, which is characterized by the appearance of a circumscribed nodule (figs 115 and 116).

The second type, which was only twice encountered in our

series of specimens is not the round, circumscribed form probably on account of the more invasive growth. The macroscopic picture of this type is a streak of tumour tissue (fig 117), usually a wide band of varying length occasionally involving a whole area (fig 118). This type of tumour reminds us of the way in which cancer of the gall bladder extends into the liver. We originally named this type of growth the diffusely growing type it would perhaps be better to speak of a locally diffuse growing type of metastases.

Both types when occurring as secondary growth start as a small tumour embolus in the liver. The *nodular type* grows out eccentrically and pushes aside the normal liver tissue as well as the local hollow structures.

None of the intrahepatic structures at the site of the nodule proved to be filled with polyvinylite in our casts. Occasionally some minor hepatic arterial branches may penetrate usually a very short distance into the periphery of the tumour nodule. In this type the larger branches curve around the tumour nodules with a definite displacement curvature in their course (both arteries and veins) (fig 116).

We did not investigate whether the structures originally present at the site of the nodule atrophied during the process of extension of the tumour during which these structures were pushed aside or became occluded (by invasion or compression). In the *diffusely growing type* however the arteries and most of the bile ducts are not occluded not even the tiny branches the portal vein branches are except for some major branches (fig 118) totally occluded.

In cancer of the gall bladder the liver is invaded on either side of the fissura media all structures being occluded except some very small hepatic arterial branches penetrating not further than into the periphery of the tumour growth. In this case the growing tumour pushes aside the major arterial branches and very many of these branches are re-discovered at the border line between tumour and normal liver tissue.

None of the structures in the vicinity of the tumour are filled however, which provides evidence of a considerable pressure exerted on these vessels, with or without occlusion from within by *thrombosis and/or invasion by tumour cells*

The practical lesson afforded by this fact is that in all types of secondary hepatic malignancies the portal vein branches are occluded. The possibility of utilizing this fact for diagnostic purposes ought to be considered. Phlebography prior to surgery, as described by Abeatici and Campi (1948), or during surgery, by injecting 35-70% diodrast into the *superior mesenteric vein*, may prove to be a valuable diagnostic procedure for ascertaining whether there are metastases in the liver (if they are large enough to become detectable in this way), where they are localized (which requires a proper knowledge of the normal anatomy) and how many metastases are present.

SUMMARY

bon nombre de jeunes condisciples future chirurgiens sont tout à fait erudits sur les moindres rameaux de la maxillaire inférieure ou sur les moindres variétés des tumeurs palmariales ou plantaires alors qu'ils possèdent des notions souvent rudimentaires sur l'anatomie de l'artère hépatique

With these words Rio Branco in 1912 introduced his truly authoritative study. Although in the forty years or so that have passed since this study was first published the various views and ideas in surgery have undergone radical changes—by no means least those concerned with the position and significance of anatomy this development has gone by imperceptibly in hepatic surgery with the result that this branch of surgery is still in almost the same condition as around 1880-1890. Thus in itself could be nothing unusual were it not that the various methods that have been developed possess disadvantages which have deterred many from applying them.

We have therefore reviewed briefly the various methods of partial liver resection that have been described together with their attendant drawbacks. We now consider that a great number of these drawbacks no longer apply in that form of surgery which takes into account the anatomical structure and special features of the liver. For this reason also the first part of this work is devoted entirely to a study of the anatomy of this organ and its internal structure.

With regard to the division of the liver it appears that from its outward appearance it can be divided not only into a right and left lobe with the attachment of the ligamentum falciforme as the dividing plane—as one usually finds it in the textbooks—but also and in our opinion much more correctly into a left, right and middle lobe as has already been suggested by Ruge and De Burlet. The middle lobe can be further subdivided into two or three lobes.

When one studies the behaviour of the structures of the Glissonian system with the help of the findings in corrosion preparations it seems that the intrahepatic flow bed of this system splits into two halves both of which are subdivided into lobes and some of these can be subdivided into segments. In comparing the areas distinguishable as such in the liver with the external subdivisions of the organ observed by Ruge and De Burlet the lobes described by these authors appear to a certain extent to correspond to those areas

We have naturally attempted to give an embryological explanation for this subdivision of the liver. Although there are definite indications that these subdivisions are indeed founded along the lines on which the intrahepatic venous systems develop, we feel at the same time that insufficient data are as yet available to support this view and that further research is advisable.

It is impossible to summarize in a few words the topographical relationship of the various structures of the Glissonian system especially since they show such great variability. We therefore refer the reader to the chapters concerned (III and IV) in which with the help of several diagrams, the most frequently occurring pictures are reproduced.

The vena hepatica system is in this respect simpler. Next to the *venae hepaticae majores* one can discern the *venae hepaticae breves*. The former are naturally of most interest to us and amongst them we can distinguish the *vena hepatica dextra*, *sinistra* and *media*. These hepatic veins lie in the interlobar fissures of the Glissonian system, i. e., the *vena hepatica dextra* in the right, the *vena hepatica sinistra* in the left interlobar fissure and the *vena hepatica media* in the median fissure.

One may wonder to what extent the intrahepatic vascular system opens perspectives in the surgery of the liver. It appears that neither the division of the Glissonian system nor that of the vena hepatica system forms in itself a satisfactory basis, yet when one combines both of these one arrives at a division into three surgical liver lobes each having its own afferent and efferent systems at both hepatic gateways.

The principles of hepatic surgery based on the morphology of the liver can be deduced easily from the preceding remarks. In this form of surgery the pivot of the method lies in the ligation at the liver hilum of the Glissonian structures in that part of the liver to be resected and of the corresponding branch of the vena hepatica at the cranial pole of the liver. It is then pointed out that the interlobar fissures dividing the lobes from each other are the indicated planes along which the resection should be made.

This method has the advantage that it partly or entirely eliminates the complications of classical hepatic surgery, viz. the chance of bleeding and cholelithiasis and—if properly applied—the chance of damage to afferent and efferent structures of the remaining part of the liver.

In considering the various surgical questions one cannot escape the problem which incisions enable the surgeon to reach the differ-

ent parts of the liver. It is self evident that the incision must depend on the nature of the resection. A number of these incisions were checked on cadavers and are discussed in Chapter IX.

Further we felt that we should consider the therapeutic consequences of hepatic surgery. We take the view that for this radical surgery, resection should be considered only for those pathological affections with which every other form of therapy will certainly or almost certainly fail. We have therefore provisionally drawn up the following list of indications for resection: primary solitary malignant growths, isolated metastatic tumours, carcinoma of the gall bladder in the initial stage of its invasion of the liver, and deep lacerations of the liver.

Finally, in the last chapter various physiological and clinical aspects connected with hepatic surgery are briefly discussed.

Many problems remain unsolved. Among the ones not yet investigated are those concerning the innervation of the liver, the role that the liver plays in the circulation of lymph, and the influence on them of possible damage during surgery. Other problems less directly concerned with the subject are just touched on.

If this work is able to render successfully some of the most important aspects of the anatomy and surgery of the liver, and to interest some investigators in the problems yet to be solved, it will have fulfilled its purpose.

- ANDE R W (1911) Large primary carcinoma of the liver *Ann Surg* 54 236
- ABDERHALDEN F (1946) *Lehrbuch der physiologischen Chemie* Benno Schwabe & Co Basel
- Abdominal and genito urinary injuries *Military Surg Manuals P III National Research Council* W B Saunders Philadelphia 1943
- AGRATICI S and CAMPI L (1931) Sur les possibilités de l'angiographie hépatique La visualisation du système portal Recherches expérimentales *Acta Radiol* 36 383
- ABEL L (1934) Primary carcinoma of the liver with report of a case successfully treated by partial hepatectomy *Brit J Surg* 21 684
- ACKERMAN F D and RHEA L J (1931) Non parasitic cysts of the liver The clinical and pathological aspects *Brit J Surg* 18 648
- AHLNSTIHL A (1896) Die Lebergeschwulste und ihre Behandlung *Arch Klin Chir* 52 902
- ALCA Y (1935) Ueber einen seltenen Fall von operativdauernd geheiltem Gallenblasen Karzinom *Zentralbl f Chir* 62 217
- ALBINI BERNARDI S (1761) *Explicatio Tabularum Bartholomaei Eustachii*
- ALBRECHT (1904) Ueber Hamartome *Verhandl Dtsch Path Ges* 7 153
- ALTMAN W A (1931) Resection of left lobe of liver for benign hemangioma *J Amer Med Ass* 146 254
- ANDERSON W A D (1948) *Pathology* C V Mosby St Louis
- ANDREWS F THOMAS W A and SCHLAFER K (1908) Newer aspects of liver disease *Surg Gyn and Obst* 47 179
- ANSCHUTZ W (1903) Ueber die Resektion der Leber *Samml Klin Vortrage A f Nr* 356-357
- ANSCHUTZ W (1907) Neue Beiträge zur Leberresektion *Arch Klin Chir* 84 335
- ARLEN I (1931) Rupture of the liver in the newborn *Med J Austr* 2 632
- ARFY C H (1947) *Developmental anatomy* 5th ed W B Saunders Philadelphia
- AURIG G SLESS H J KOTHE W and SCHOTZ O (1934) Zur Kontrastdarstellung des Pfortaderkreislaufes nach perkutaner transperitonealer Milzpunktion *Fortschr Geb der Kontgenstr* 81 1
- ACARAY M (1897) Sur la résection du foie chez l'homme et les animaux *Rev Chir* 27 645
- BARCLAY A F FRANKLIN K J and IRICHARD M M I (1916) *The foetal circulation and cardiovascular system and the changes they undergo at birth* Blackwell Scientific Publ Oxford
- BARIATTI R and DAVRADI A (1937) Ligation of the hepatic artery Possibility of modifying the sequelae *Arch Ital Chir* 75 16
- BARON A (1910) Blutlose Leberoperationen *Zentralbl f Chir* 37 1537

- BAX H R and SCHALM L (1934) Nieuwe mogelijkheden op het gebied der leverchirurgie *Ned Tijdschr v Geneesk* 98 3140
- BENSON C D and FERNBERTH G C (1942) Surgical excision of primary tumour of liver (hamartoma) in infant 7 months old with recovery *Surgery* 12 831
- BENZ F J BACCENSTOSS A H and WOLLATZER E F (1932) Atrophy of the left lobe of the liver *Arch Path* 53 315
- BENZ F J and BACCENSTOSS A H (1933) Focal cirrhosis of the liver. Its relation to the so-called hamartoma (adenoma benign hepatoma) *Cancer* 6 743
- BERENCZY G VON and WOLFF K VON (1924) Ueber die Verbreitung des Carcinoms auf Grund von 19 908 Sektionen des St. Stephanshospitals in Budapest *Zeitschr f Krebsforsch* 21 109
- BERESNECOWSKI N (1914) Ueber die Anwendung des isolierten Netzes zur Stillung der Leberblutung. Experimenteller Untersuchung *Arch Klin Chir* 104 287
- BERGMANN F VON (1893) Zur Casuistik der Leber Chirurgie *Arch Klin Chir* 46 393
- BERMAN C (1931) *Primary carcinoma of the liver* H K Lewis & Co Ltd London
- BLALOCK A (1924) Statistical study of eighthundred and eighty-eight cases of biliary tract disease *Bull J Hopkins Hosp* 35 391
- BOGLE J H (1948) Anomaly of the right hepatic artery hitherto undescribed and its surgical significance *The Brooklyn Hosp Journ* 1 209
- BOLJARSKI N (1910) Ueber Lebererletzungen in klinischer und experimenteller Hinsicht unter besonderer Berücksichtigung der isolierten Netzplastik *Arch Klin Chir* 93 507
- BOLLMAN J L (1943) The influence of diet on the resistance to experimental hepatitis *Surg Clin N Amer* (Aug) 23 1102
- BONNE C (1932) Het primaire levercarcinoom *Ned Tijdschr v Geneesk* 76 438
- BONNE C (1933) Verschillen in de kankerstatistieken te Medan en Batavia *Geneesk Tijdschr Ned Ind* 73 1440
- BOURNE W (1939) Factors determining selection and administration of anaesthetics *Surg Gyn and Obst* 68 520
- BOURNE W and ROSENTHAL (1928) The effects of anesthetics on liver function *J Amer Med Ass* 90 377
- BOURNE W BRUGER M and DREYER N III (1930) The effect of sodium amytal on liverfunction *Surg Gyn and Obst* 51 356
- BOURNE W and RAGINSKI B B (1934) The effects of cyclopropane on the normal and impaired liver *Canad Med Ass J* 31 50
- BOYCE F F (1935) The so called liver death syndrome in surgery *Surg Gyn and Obst* 61 1
- BOYCE F F McFERRIDGE E M and VEAL J R (1936) A clinical and experimental study of the so-called liver death *Surg Gyn and Obst* 63 43

- ✓ DE BURLET H M (1910) Zwei Fälle von abnormer Spaltbildung an der menschlichen Leber *Morph Jahrb* 42 476
- DEELMAN H T (1940) *Leerboek der pathologische anatomie* Traven F Bohn Haarlem
- DE JOSSELYN DE JONG, R (1912) Ueber die Folgen der Thrombose im Gebiete des Pfortadersystems *Mitt a d Grenzgeb Med u Chir* 24 160
- DFLORME E J (1951) Arterial perfusion of the liver in shock *Lancet* i 259
- DENTON R W and IVY A C (1948) Effect of feeding liver on the rate of regeneration of the liver in partially hepatectomized rats *Amer J Physiol* 152 460
- DESCOMPS P and LALAUBIE J DE (1910) Les vaisseaux sanguins et les voies biliaires dans le hile du foie *Bull et Mém de la Soc Anat de Paris* 85 323
- DEVÉ F (1913) Les localisations de l'échinococcose primitive chez l'homme Nécessité d'une révision des statistiques *C R Soc Biol* 74 735
- DE VOS J F (1954) *Personal communication*
- DICK H M (1928) Stream lines in the portal vein their influence on the selective distribution of blood in the liver *Edinb Med J* 35 533
- DOCK W (1947) The clinical significance of some peculiarities of the circulation in the kidneys lungs and heart *New Engl J Med* 236 773
- DONOVAN E J and SANTULLI J (1946) Resection of the left lobe of the liver for mesenchymoma *Ann Surg* 124 90
- DOUGLASS H E BAGGENSTOSS A H and HOLLINSHEAD W H (1950) The anatomy of the portal vein and its tributaries *Surg Gyn and Obst* 91 562
- DU BOULAY G H and GREEN H (1954) Portal venography in Banti's disease *Brit J Radiol* 27 423
- DUMPHY J E HARRISON J H and MERRILL J P (1951) The nature of hepatic failure complicating surgery of the gall bladder and bile ducts *Postgrad Med Surg* 176-178
- DUVERNOY G L (1835) Etude sur le foie *Ann Sciences Naturelles II* 101 Zool (I c F Meyer)
- EDLER L VON (1887) Die traumatische Verletzungen der parenchymatösen Unterleibsorgane (Leber Milz, Pankreas Nieren) *Arch Klin Chir* 34 173
- EGGEL H (1901) Ueber das primäre Karzinom der Leber *Beitr Path Anat* 30 505
- ✓ ELIAS H (1953) Functional morphology of the liver *Research Ser. Med* 37 26
- ✓ ELIAS H and PETTY D (1952) Gross anatomy of the blood vessels and ducts within the human liver *Im J Anat* 90 59
- ELLENBERGER and BAUM (1908) *Handbuch der vergleichende Anatomie der Hausaugetiere* 12 Aufl (I c MEYER F and MERMANN F V V M)

- ELLENBERGER M and OSTERMAN K F (1931) Role of shock in production of central liver cell necrosis *Amer J Med* 11 170
- ELKHJIM C A and KREHL W H (1937) Imbalance and dietary interrelationships in nutrition *J Amer Med Ass* 135 279
- EPINGER H (1937) *Die Leberkrankheiten Allgemeine und spezielle Pathologie und Therapie der Leber* J Springer Wien
- FISHER H J and HIRSCHUTZ B (1932) Hemobilia cholecystitis and gastro-intestinal bleeding in rupture of the liver *J Amer Med Ass* 149 1132
- FISCH C I (1936) *Starlings principles of human physiology* J & A Churchill London
- FISCH H M (1912) The development of the vascular system *Keibel and Mall Human Embryol II*
- EWING J (1928) *Neoplastic diseases* 3d ed W B Saunders Philadelphia
- FAIRCCHILD I F (1931) Serious intra abdominal trauma without evidence of violence *Surg Gyn and Obst* 52 767
- FICARRA B J (1930) Surgical death due to hepatic exhaustion *Amer J Surg* 80 104
- FINE J (1932) Relation of bacterial action to development of state of irreversibility in shock In *Shock and circulatory homeostasis Transactions of the 2d Conf on shock sponsored by Josiah Macy (Jr) Found*
- FINE J SELIGMAN A M and FRANK H A (1947) On the specific role of the liver in haemorrhagic shock *Ann Surg* 126 1002
- FINE J and ZWEIFACH B W (1932) Mechanism by which bacteria exert a deleterious effect on the circulation In *Shock and circulatory homeostasis Transactions of the 2d Conf on Shock sponsored by Josiah Macy (Jr) Found*
- FINSTERER H (1910) Ein geheilter Fall von Leberschuss *Wiener Klin Wochenschr* 23 632
- FINSTERER H (1911) Zur Diagnose der Leberverletzungen *Arch Klin Chir* 95 376
- FINSTERER H (1930) Das Karzinom der Gallenblase *Med Klin* 28 432
- FITTS W J (1932) The antibiotics and liver injury *Surgery* 30 475
- FLETCHER H A (1949) Spontaneous disappearance of apparently secondary growth in the liver *Brit Med J* 46 794
- FLOWER W H (1872) *Le MEYER F*
- FLOWERS M W A (1872) Sur la disposition et la nomenclature des lobes du foie chez les mammifères *J Zool* 1 40
- FORBES E B SWIFT R W FILLIOT R F and JAMES W H (1946) Relationship of fat to economy of food utilization *J Nutrition* 31 203
- FRAZER D RAFFAPORT A M VUYLSTEKE C A and COLWELL (Jr) A R (1931) Effects of the ligation of the hepatic artery in dogs *Surgery* 30 624
- FRERICHS F T (1861) A clinical treatise on disease of the liver *The New Sydenham Soc London* 21 512

- FRIEDMAN L W FRANK H A and FINE J (1931) Portal circulation in experimental haemorrhagic shock *Ann Surg* 134 70
- FURTWANGLER A (1927) Diffuse Rindennekrose beider Nieren nach Leberterruptur *Krankheitsforsch* 4 349
- GALENUS (1550) *De usu partium corporis humani* G Rouillium Lugduni
- GANS H (1934) De anatomie van de normale en carcinomateuze lever en haar betekenis voor chirurgie en diagnostiek *Voordracht St Canisius ziekenhuis Nijmegen* (April)
- GANS H (1934) Over de hemi hepatectomie *Voordracht Ned Ver Heel kunde* (Sept) *Ned Tijdsch v Geneesk* 99 1693 (1935)
- GANS H (1935) De lever als een gepaard gelobd en gesegmenteerd orgaan *Voordracht Ned Anat Ver* (Febr)
- GANS H (1935) On the anatomy of the liver and its repercussions on surgery Preliminary report *Arch Chir Neerl* Nr 2
- GARLOCK J H (1946) Combined abdomino thoracic approach for carcinoma of cardia and lower esophagus *Surg Gyn and Obst* 83 731
- GARRE (1907) On resection of the liver *Surg Gyn and Obst* 5 331
- GERDING W J POPP M F and MARTINEAU P C (1931) Hamartomatous cholangio-hepatomas *J Amer Med Ass* 143 821
- GERSHBEIN L L and ELIAS H (1934) Observation on the anatomy of the rat liver *Anat Rec* 120 85
- GILFILLAN R S (1930) Anatomical study of the portal vein and its main branches *Arch Surg* 61 449
- GILLMAN J and GILLMAN F (1948) Anoxia and the liver with special reference to shock and chronic malnutrition *S Afr J Med Sc* 13 11
- GIRARD J (1830) *Traité d'anatomie vétérinaire* T c F MEYER
- CLENARD and SIRAUD (1895) Sur les modifications de l'aspect physiques et des rapports du foie cadavérique par les injections aqueuses dans les veines de ces organes *Lyon Med* 89, 902
- GLENN F and HAYS D M (1930) The causes of death following biliary tract surgery for non malignant disease *Surg Gyn and Obst* 91 283
- GLISSON F (1654) *Anatomia hepatis* O Pullein London
- GLUCK TH (1883) Ueber Extirpation von Organen *Arch Klin Chir* 28 604
- GLUCK TH (1884) Ueber die Bedeutung physiologisch chirurgischer Experimente an der Leber *Arch Klin Chir* 29 139
- GORDON TAYLOR G (1940) The problems of surgery in total war *Surg Gyn and Obst* 74 375
- GRAHAM E A (1931) The prevention of carcinoma of the gall bladder *Ann Surg* 93 317
- GRAHAM R R and CANNELL D (1933) Accidental ligation of the hepatic artery *Brit J Surg* 20 566
- GRAY H K and SHARIF W S (1941) Carcinoma of the gall bladder extrahepatic bile ducts and the major duodenal papilla *Surg Clin A Amer* 21 1117

- CRENS H D (1933) Shock and circulatory homeostasis *Trans 2d Conf sponsored by Josiah Macy (Jr)* New York Corlies Macy & Co Inc NY
- CRINDLAY J H and BOHLMAN J L (1932) Regeneration of the liver in the dog after partial hepatectomy. Role of the venous circulation *Surg Clin and Obst* 93 491
- CRUFWALD I (1919) Degenerative changes in the right half of the liver resulting from intra uterine anoxia *Im J Clin Path* 19 801
- CURD I A, LARSEN H M and RAYDIN I S (1918) Composition of the regenerating liver after partial hepatectomy in normal and protein depleted rats *Im J Physiol* 152 11
- CURLT F F (1844) *Handbuch der vergleichende Anatomie der Haussaugetiere* 3 Aufl Jc Meyer J
- HABERER H von (1903) Experimentelle Unterbindung der Leberarterie *Arch Klin Chir* 78 357
- HALLER A von (1764) *Elementa physiologiae corporis humani* T VII and VIII Bern
- HAMILTON W J, BOYD J D and MOSSMAN H W (1932) *Human embryology* 2nd ed W Heffer Cambridge
- HAMILTON BAILEY (1911) *Surgery of modern warfare* E & S Livingstone Edinburgh
- HAMNER I (1928) Maximale reductie van één leverkwab *Ned Tijdschr Geneesk* 72 226
- HASOT and CILBERT (1888) *Etudes sur les maladies du foye* Asselin et Houssau Paris
- HARPER F R (1917) Thoracic-abdominal approach to the upper abdomen *Surg Gyn and Obst* 84 331
- HARTL F (1932) Anatomische Untersuchungen von Leber, Niere und Pankreas bei postoperativen Todesfällen (Zugleich ein Beitrag zum Hepatorenalen Syndrom) *Arch Klin Chir* 271 121
- HARTMAN (1901) *Chirurgie gastro-intestinale* Paris
- HARTROFT W S (1918) Some histological aspects of renal and hepatic acholinopathy. In *Liver injury* *Trans 6th Conf Josiah Macy (Jr) Found* (May) p 41-48
- HASTINGS-JAMES R (1919) Malignant haemangio-endothelioma (haemangioblastoma) of the liver *J Path Bact* 61 49
- HEALEY J E (1934) Clinical anatomic aspects of radical hepatic surgery *J Int Coll Surg* 22 542
- HEALEY J E and SCHROY P (1933) The anatomy of the bile ducts within the human liver. An analysis of the prevailing patterns of branching and their major variations *Arch Surg* 66 399
- HEALEY J E, SCHROY P and SORENSSEN R (1933) The intrahepatic distribution of the hepatic artery in man *J Int Coll Surg* 20 133
- HELWIG F C and ORR T G (1932) Traumatic liver necrosis with extensive creatinine retention and high grade nephrosis *Arch Surg* 74 136

- HELVIG F C and SCHUTZ C B (1932) Liver kidney syndrome Clinical pathological and experimental studies *Surg Gyn and Obst* 55 50
- HENDRICK J G (1948) Haemangioma of the liver causing death in a newborn infant *J Pediatr* 32 309
- HENLE J (1868) *Handbuch der systematischen Anatomie des Menschen* III Handbuch der Gefasslehre Braunschweig
- HENSCHEN (1932) Die Bedeutung der Leber in der Chirurgie *Arch Klin Chir* 173 488
- HERBEN J (1948) Primary carcinoma of the liver *Acta Chir Belg* 5 509
- HEYD C G (1924) Hepatitis a condition associated with gall bladder disease *Surg Gyn and Obst* 39 66
- HEYD C G (1924) The liver and its relation to chronic abdominal infection *Ann Surg* 79 55
- HEYD C G (1933) Editorial *Surg Gyn and Obst* 57 407
- HIGGINS G M MANN F C and PRIESTLY J F (1939) Experimental pathology of the liver *Arch Pathol* 14 491
- HILDEBRAND VON (1907) Ueber die Methode durch Einbringen von Schattengebenden Flüssigkeiten Hohlorgane des Körpers im Röntgenogramm sichtbar zu machen *Fortschr a d Geb d Röntgenstr* 11 207
- HIMSWORTH H P (1946) Protein metabolism in relation to disease *Proc Roy Soc Med* 40 27
- HIMSWORTH H P and GLYNN, L E (1944) Toxicopathic and trophopathic hepatitis *Lancet* 1 457
- HIPPEL H (1910) Zur Kenntnis der Mischgeschwulste der Leber *Arch f Pathol Anat* 201-306
- HIS (1895) *Anatomische Nomenklatur* Basel L C F MEYER
- HJORTSJO C H (1948) Die Anatomie der intrahepatischen Gallengänge beim Menschen mittels Röntgen und Injektionstechnik studiert *Annl J Fysogr Sällskapet's Handl A F* 59 1
- HJORTSJO C H (1951) The topography of the intrahepatic duct system *Acta Anat* 11 599
- HONIGLAND J C RAYDIN and VARS
- HOLMAN L (1950) Surgical approach to upper abdomen through incision paralleling the costal margin with excision of costal cartilage opposite the fifth intercostal space *Am J Surg* 90 464
- HUMPHREYS G H (1946) An approach to resection of the esophagus and gastric cardia *Ann Surg* 124 288
- HYDEN H (1950) Ueber die Frequenz des Gallenblasen Karzinoms *Zentralbl f Chir* p 674
- HYRTL J (1873) *Die Corrosions Anatomie und ihre Ergebnisse* Wilhelm Braumüller Wien
- HILLE J E W BONGERSMA L D VANDERKLAAM C J DE LANCE (Jr) DE VAN OORDT G J RAVEN CHIR SLIJPER F J HINDERSEN N and VORSTMAN A G (1947) *Leerboek der vergelijkende ontleedkunde van de vertebraten* N V Oosthoek's Uitg Mij Utrecht

ITINWORTH C I W (1933) Carcinoma of the gall bladder *Brit J Surg* 23 4
 IWAKI K (1914) Ueber das primäre Sarkom der Gallenblase *Arch Klin Chir* 104 84

JACQUIN I (1913) Leber Blutstillung bei Leberwunden durch gestielte und freie Netzlappen *Arch Klin Chir* 102 302
 JACQUIN I and WOHLFARTH J (1913) Eine neue Methode zur Stillung parenchymatöser Blutungen *Arch Klin Chir* 106 104

JANFELSON I R (1937) Clinical aspects of primary carcinoma of the gall bladder *New Engl J Med* 217 83
 JOHNSON D H (1931) The effect of haemorrhage and hypotension on the liver bloodflow *J Physiol* 126 413

JOHNSTON I A and ANSON B J (1932) Variations in the formation and vascular relationships of the bile ducts *Surg Gyn and Obst* 94 669
 JONES C J (1930) Carcinoma of the gall bladder A clinical and pathologic analysis of 50 cases *Ann Surg* 132 110

JUDD F S and BALMARTER C J (1929) Malignant lesions of the gall bladder *Arch Int Med* 44 733
 JUDD F S and CRAY H K (1932) Carcinoma of the gall bladder and bile ducts *Surg Gyn and Obst* 55 308

KALFMAN E (1932) *Spezielle pathologische Anatomie* 9 und 10 Aufl Walter de Gruyter & Co Berlin & Leipzig
 KAY S and TALBERT I C (1930) Adenoma of the liver mixed type (Hamartoma) Report of two cases *Cancer* 3 307

KEEN W W (1899) Report of a case of resection of the liver for the removal of a neoplasm with a table of 76 cases of resection of the liver for hepatic tumours *Ann Surg* 30 267

KFIR H (1903) *Technik der Gallensternoperationen* München
 KIERNAN F (1833) The anatomy and physiology of the liver *Philos Tr Roy Soc London* 133 711

KLEINSCHMIDT O (1913) Experimentelle Untersuchungen über Luftembolie *Arch Klin Chir* 106 82
 KNEELY M H (1949) The liver lobule In *Liver Injury Tr 8th Conf Josiah Macy (Jr) Found* p 9-13 New York

LADD W F and GROSS R E (1941) *Abdominal surgery of infancy and childhood* W B Saunders Philadelphia London
 LAM C R (1940) The present status of carcinoma of the gall bladder *Ann Surg* 111 433

LANGENBLICH (1897) *Chirurgie de Leber und Gallenblase* Deutsche Chirurgie Stuttgart
 LEE E S (1942) Large solitary bile-cell fibro-adenoma of the liver *Proc Roy Soc Med* 36 33

LEFÈVRE H (1949) L'hépatectomie gauche *Revue Française* 243-252

- LEGER I. ABEATICI S and CAMPI L. (1931) Phlébographie portale par injection splénique interparenchymateuse *Mem Inst Chir* 7, 586
- LEGER I. ALBOT G and ARVAY M (1931) La phlébographie portale dans l'exploration des affections hépato spléniques *Presse Méd* 59 1230
- LEGER I. ALBOT G. OLDOT J and ZEROLO J (1931) Effet de la ligature de l'artère hépatique sur le système portale *Presse Méd* 59 1230
- LEWITZE E (1926) Gallensteine und Gallenblasencarcinom *Beitr Klin Chir* 137 38
- LEYH F A (1850) *Handbuch der Anatomie der Haustiere* Lc F MEYER
- LICHTENSTEIN, G M and TAYLORBAUM W (1940) Carcinoma of the gall bladder *Ann Surg* 111 411
- LICHTMAN S S (1933) *Diseases of the liver gall bladder and bile ducts* 3d ed Henry Kimpton London
- LIEBOW A A. HALEY M R. LINDSKOG G E and BLOOMER W I (1947) Plastic demonstrations of pulmonary pathology *Bull Int Ass Med Museums* 28 116
- LIEBOW E (1915) Leber Bauchschüsse insbesondere über Schussverletzungen der Leber *Arch Klin Chir* 107 509
- LOCALIO S A and SALTZ N J (1930) Regeneration of the liver following massive destruction as a result of trauma. Report of a case and correlation with experimental literature *Surgery* 27 282
- LORTAT JACOB J L and ROBERT H G (1931) Hépatectomie droite réglée *Presse Méd* 60 549
- LOWE C R and OVIARY D C (1931) An evaluation of rigid dietary sodium restriction in the management of ascites in cirrhosis of the liver *Ann Int Med* 34 1396
- MALL F P (1906) A study of the structural unit of the liver *Amer J Anat* 5 227
- MALPIGHI (1669) Lc VON HABERER
- MALTSCHEFF G V (1930) A rupture of the liver following delivery *Akush Ginek* 6 54
- MANN I C (1934) Hepatic function in relation to hepatic pathology *Ann Int Med* 8 432
- MANN I C (1941) Liver and medical progress *J Amer Med Ass* 117 1277
- MANN F C (1912) The circulation of the liver *Quart Bull Indiana Uni* 4 43
- MANN F C (1913) The gastro-intestinal tract and the liver *J Amer Med Ass* 119 720
- MANN F C (1916) The portal circulation and restoration of the liver after partial removal *Surgery* 8 225
- MANN F C. FISHBACH F C. GRAY J C and CREECH C I (1931) Experimental pathology of the liver *Arch Pathol* 12 787

- MARROWITZ (1932) The hepatic artery *Surg Gyn and Obst* 95 614
- MARTIN L. (1920) Röntgenologische Studien zur arteriellen Gefäßversorgung in der Leber *Arch Klin Chir* 114 1001
- MARTIN (Jr) J D (1911) Wounds of the liver *Ann Surg* 123 756
- MASON I C and DAVENPORT I C (1923) Study in tissue autolysis in liver *J Clin Lab Med* 10 622
- MASON R L and ZINTZ H A (1917) *Pre-operative and post-operative treatment* W B Saunders Philadelphia London
- MASSÉ L and DUBOURG G (1930) La cholecysto-hepatectomie dans les cancers de la vésicule *Arch Mal del App Dig* 39 261
- MAYER L. (1872) *Verletzungen der Leber und Gallenblase* R Oldenburg München
- MCCORMACK H and HOWARD I S (1912) Severe trauma to the liver with hepatorenal syndrome *Ann Surg* 116 223
- MCDONALD R I THOMAS W A and ANDREWS E (1928) *1c An*
- DEWIS I THOMAS W A and SCHLEICHER A (1927) The bilaterality of the liver *Arch Surg* 13 389
- McLADDOFF A H and GUNNING A S (1927) The bilaterality of the liver *Arch Surg* 13 389
- McRae R W (1933) Unusual tumour (malignant adenoma) of the liver of a baby *Ann J Surg* 28 272
- MEAL E. C (1931) The effect of anaesthesia upon the blood sugar content *Surg Gyn and Obst* 53 329
- MEKIE F C. and MILLER H (1929) The effect of anaesthesia operation and certain other factors of glycaemia *Brit Med J* 1 244
- MEYER F (1911) *Terminologie und Morphologie der Säugetierleber* Nest verschillende buikorganen ook intrahepatisch langs bepaalde wegen? *Dus Amsterdam* Scheltema & Holkema Amsterdam
- MEYER F (1911) *Terminologie und Morphologie der Säugetierleber* Nest verschillende buikorganen ook intrahepatisch langs bepaalde wegen? *Dus Amsterdam* Scheltema & Holkema Amsterdam
- MICHELIS W A (1946) Variations in blood supply of liver gall bladder stomach duodenum and pancreas *Int Rec* 94 481
- MIKAL S and PAIZEN G W (1930) Morbidity and mortality in ruptured liver *Surgery* 27 570
- MOLLER W (1936) Leberresektion wegen Krebsmetastasen 6-jährige lokale Rezidivfreiheit *Acta Chir Scand* 78 103
- MORRISON L M (1946) The response of cirrhosis of the liver to an intensive combined therapy *Ann Int Med* 24 463
- MORRISON L M (1947) New methods of therapy in cirrhosis of the liver *J Amer Med Ass* 134 673
- MURRAY J F and KIMMEL S A (1949) Chiani syndrome Report of a case *S Afr Med J* 23 831
- MUSSEY (1889) Primary cancer of the gall bladder *Ann Surg* 93 317
- NARATH A (1909) Ueber die Unterbindung der Arteria Hepatica *Beitr Klin Chir* 45 204
- NARATH A (1915) Die arterio-venose Anastomosis an der Pfortader als

LITERATURE

- Mittel zur Verhütung der Lebernekrose nach Unterbindung der Arteria
Hepatica *Zentralbl Chir* 42 1
- VASSE (1894) Ueber Experimente an der Leber und den Gallenweg n
Arch Klin Chir 48 883
- NAU M P (1903) Le développement du foie *Bull et Mem Soc Anat
Paris* 2 100
- NEWMAN E GROSSMAN M I and IVEY A C (1949) Effect of diet on liver
regeneration in partial hepatectomized rats *Amer J Physiol* 133 221
- NOEGENRATH E (1854) Geburtshinderniss in Folge eines Lebercarci
noms beim Neugeborenen *Dtsch Klin* 6 496
- NOETZEL W (1910) Experimentelle Untersuchungen zur Gallenblasen
perforationsperitonitis *Arch Klin Chir* 93 116
- NORMAN O (1931) Studies on the hepatic ducts in cholangiography
Acta Radiol Suppl 84 1
- NYSTROM T G (1932) Liver resections in primary malignant hepatoma
Acta Chir Scand 103 241
- OEHLCKER F (1932) Leberveränderungen bei Verschluss des linken
Astes der Pfortader *Arch Klin Chir* 173 663
- OIDTMANN (1920) Partiele leverresectie voor levergezweel *Ned Tijdschr
Geneesk* 64 2617
- O'NEILL J N (1941) Traumatic rupture of the liver *Calif & West Med
54* 68
- O'NEILL SHERMAN W (1934) Abdominal injuries *Surg Gyn and O
58* 307
- ORR T G and HELWIG F C (1939) Liver trauma and hepatorei
syndrome *Ann Surg* 110 682
- OTIS I c BOLJARSKI
- PAREIRA M D and PROBSTEN J G (1949) Glucose assimilation during
anaesthesia and surgery *Ann Surg* 129 463
- PIHR E (1908) II Internat Chirurgenkongress Brussel *Zentralbl f
Chir* 33 1341
- PAYR E and MARTINI A (1903) Experimentelle und klinische Beiträge
zur Lebernaht und Leberresektion (Magnesiumplattennaht) *Arch Klin
Chir* 77 962
- PRICK M F and GROVER R F (1932) Cardiovascular responses to acute
ligation of portal vein *Arch Surg* 64 663
- PHILLIP P W (1907) Ueber Krebsbildung im Kindesalter *Z Krebs
forsch* 33 6
- PHILLIPS R KARLOVSKY D A HAMILTON L D and NICKSON J J
(1934) Roentgentherapy of hepatic metastases *Amer J Roentgenol and
Radiumther* (May) 826
- PICKRELL A L and CLAY R C (1944) Lobectomy of the liver Report
of three cases *Arch Surg* 118 266
- POLITZER C (1933) Die symmetrische Form der Leber bei der Hepatom

- phalon und die Ursachen der normalen Asymmetrie der Leber. *Wien Klin Wochschr* 6, 800
- IONICKI J (1879) Experimentelle Beiträge zur Pathologie der Leber. *Arch f Path Anat u Physiol* 118, 49
- QUATREFAUM J A (1903) Massive resection of the liver. *Ann Surg* 137, 8
- QUEL J L (1909) De l'opération radicale dans le cancer des voies biliaires. *Pr. Chir* 39, 243
- RACINSKI B B See BOURNE H and RACINSKI B B
- RANWITZOFF I C CARRE
- RAPPORT A and HROMADA J (1930) A contribution to the surgical significance of aberrant hepatic ducts. *Ann Surg* 132, 233
- RAPPAPORT A M BOROWY J LOUBOFF W M and LOTTO W V (1934) Subdivision of hexagonal liver lobules into a structural and functional unit. Role in hepatic physiology and pathology. *Anat Rec* 119, 11
- RAVDIN I S (1929) Some aspects of carbohydrate metabolism in hepatic disease. *J Amer Med Ass* 93, 1193
- RAVDIN I S (1938) Some observations on normal and pathologic liver functions. *Amer J Surg* 40, 171
- RAVDIN I S (1939) Some recent advances in surgical therapeutics. *Ann Surg* 109, 321
- RAVDIN I S (1940) Hypoproteinemia and its relation to surgical problems. *Ann Surg* 112, 576
- RAVDIN I S (1951) Some factors concerned with liver injury. *West J Surg* 59, 531
- RAVDIN I S and VARS H M (1950) Further studies on factors influencing liver injury and liver repair. *Ann Surg* 132, 362
- RAVEN R W (1949) Partial hepatectomy. *Brit J Surg* 36, 380
- REY H (1888) Beiträge zur Morphologie der Säugetierleber. *Morph Jahrb* 14, 317
- RIBBERT H (1909) Das maligne Adenom der Leber. *Dtsch Med Wochschr* 35, 1607
- RIEDFL (1911) Zur Diagnose und Therapie des Gallenblasen Karzinoms. *Munch Med Wochschr* 58, 1337
- RIO BRANCO P DO (da Silva Paranhos) (1912) *Essai sur l'anatomie et la médecine opératoire du tronc coelomique et ses branches de l'artère hépatique en particulier*. G. Steinheil Paris
- ROLLESTON (1861) On the homologues of the lobes of the liver in mammals. *Rapport of the Brit Ass* p 174
- RUDSTROM P (1931) Hemobilia in malignant tumours of the liver. *Acta Chir Scand* 101, 243
- RLCE E (1908) Beiträge zur chirurgischen Anatomie der grossen Gallenwege. *Arch Ann Chir* 87, 42

- RUGE G (1907) Die Leber des Menschen *Morph Jahrb* 37 397 and 614
- RUGE G (1911) Ibidem *Morph Jahrb* 42 361
- RUGE G (1913) Leber mit abgespaltenem rechten Seitenlappen *Morph Jahrb* 46 293
- RUGE G (1913) Abweichungen am linken Lappen der menschlichen Leber *Morph Jahrb* 45 409
- RUGE G (1919) Spaltung des linken Lappens einer menschlichen Leber in einem Stamm und Seitenlappen *Morph Jahrb* 50 345
- SABOURIN C (1888) *Recherches sur l'anatomie normale et pathologique de la glande biliaire de l'homme* Paris
- SAINBURG F P and GARLOCK J H (1948) Carcinoma of the gall bladder *Surgery* 23 201
- SANDERS G B, MCGUIRE H CH and MOORE (JR) R H (1949) Massive rupture of the liver *Amer J Surg* 78 699
- SCHALM L (1951) The use of the regenerative power and reserve capacity of the liver in bile duct operations *Arch Chir Neerl* 3 322
- SCHALM L (1951) Critische beschouwingen over de waarde van het moderne dieet bij ziekten van het leverparenchym *Loedine* 12 401
- SCHALM L (1952) Het regeneratievermogen en de reservecapaciteit van de lever en het mogelijk nut daarvan voor de chirurgie der galwegen *Acta Clin Belg* 7 156
- SCHIFFLINO J W (1934) Een bijzonder geval van aangeboren multiple gezwollen in de lever (hamartomen) bij een kind van vier maanden *Ned Tydschr v Geneesk* 3 3566
- SCHMIDT H and GUTTMAN (1954) Röntgenuntersuchungen über die Verzweigung der grossen intrahepatischen Gallenwege *Anat Anz* 100 277
- SCHNEFELDORF J G and ORR T G (1942) The effect of distention of the small intestine, anoxemia and oxygen therapy upon the flow of bile and urine in the dog. Relationship to the hepato-renal syndrome *Surg Gyn and Obst* 74 446
- SCHORNAGEL H E and STRALB M (1954) Over het voorkomen van galblaascarcinoom. In *Verde Jaarb v Kankeronderz. en Kankerbestrijding in Nederl* J H de Bussy, Amsterdam
- SCHROEDER W E (1906) The progress of liverhaemostasis. Reports of cases *Surg Gyn and Obst* 2 52
- SCHROEDER VAN DER KOLK, I C DEFFLMAN
- SCOTT J V (1951) Use of absorbable gelatine sponge and primary suture in traumatic rupture of the liver *Amer J Surg* 81 31
- SEGALL H N (1953) An experimental investigation of the blood and bile channels of the liver *Surg Gyn and Obst* 37 152
- SÉNÉQUE J and AUROUSSEAL R (1950) Des possibilités de l'hépatectomie dans les traitements des tumeurs du foie *J de Chir* 66 77
- SÉNÉQUE J, ROLX M, CHATTELIN CH and HULLENAARD I (1952) Sur 1 cas d'hépatectomie typique gauche réglée pour kystes hydatiques

- multiples de la partie gauche du foie *Mém de l'Acad de Chir* 78 728
 SPARSHAW R D (1939) Physiological and pathological responses of
 the blood vessels of the liver *Quart J Med* 10 131
 35 77
 SARKIS H (1901) Contribution à l'étude de la circulation du sang porte
 dans le foie et des localisations lobaires hépatiques *J de Méd Bordeaux*
 31 271 291 312
 SHALLOW J I and WANDER (Jr) I B (1937) Primary fibrosarcoma of
 the liver *Amer Surg* 125 139
 SHRYVAN H L (1930) An embryonic tumour of the liver containing
 striated muscle *J Path Bact* 33 251
 SHRYVER W (1911) Cholelithiasis and partial hepatectomy for
 carcinoma of the gall bladder with local liver extension *Surgey* 22 48
 SHRYVER F J KERRY A R IFF I F LOWELL A T and ADAMS
 B J (1939) The rôle of the liver in detoxication of thiopental (pentothal) by man *Anaesthesia* 10 421
 SHORR I (1930) Recent findings concerning the rôle of the liver and
 kidney in circulatory homeostasis *Jr Liver injury Tr 8th Conf* 46 11
 SHORR I (1930) *Forum* p 60
 SHORR I and SWANSON B W and LUCIFORT R F (1943) On the
 occurrences sites and modes of origin and destruction of principles
 affecting compensatory vascular mechanisms in experimental shock
Science 102 389
 SHUMAKER (Jr) H B (1932) Hemangioma of the liver *Surgey* 11 209
 SIMMONS M (1891) Die knotige Hyperplasie und das Adenom der Leber
Dtsch Arch Klin Med 33 323
 SKRIBANSKY (1896) *Le WABST*
 SHAPPEL I and SCHWARTZ I I (1931) Transfusion-transfusion in
 hepato-renal syndrome *Ann Int Med* 34 692
 SYDER J M (1936) Surgical management of war wounds of the abdomen
Amer J Surg 12 331
 SUIJERS I I and STRAUB M (1922) Over het primair levercarcinoom
 in de tropen naar aanleiding van 17 waargenomen gevallen *Geneesk
 Tijdschr Ned Ind* 62 3
 SOLOMON H A and KATZ S J (1930) Twenty six years of survival
 following carcinoma of sigmoid with prolonged liver metastasis *J
 Amer Med Ass* 144 221
 SPICELUS A (1622) *De corporis humani fibris* Frankfurt 1c MEYER F
 STATE D RALPH R and MILLER J J (1931) The experimental pro-
 duction of the hepato-renal syndrome in dogs *Postgrad Med Surg Surg
 Forum Amer Coll Surg* p 171
 STEINER M M (1938) Primary carcinoma of the liver in childhood
 report of two cases with a critical review of the literature *Amer J
 Childh Dis* 55 807
 STEPIANSON G W (1932) Experimental pathology of the liver *Arch
 Path* 14 484

- RUGE G (1907) Die Leber des Menschen *Morph Jahrb* 37 397 and 614
- RUGE G (1911) Ibidem *Morph Jahrb* 42 361
- RUGE G (1913) Leber mit abgespaltenem rechten Seitenlappen *Morph Jahrb* 46 293
- RUGE G (1913) Abweichungen am linken Lappen der menschlichen Leber *Morph Jahrb* 45 409
- RUGE G (1919) Spaltung des linken Lappens einer menschlichen Leber in einem Stamm und Seitenlappen *Morph Jahrb* 50 345
- SABOURIN C (1888) *Recherches sur l'anatomie normale et pathologique de la glande biliaire de l'homme* Paris
- SAINBURG F P and GARLOCK J H (1948) Carcinoma of the gall bladder *Surgery* 23 201
- SANDERS G B, MCGUIRE H CH and MOORE (Jr) R H (1949) Massive rupture of the liver *Amer J Surg* 78 699
- SCHALM L (1951) The use of the regenerative power and reserve capacity of the liver in bile duct operations *Arch Chir Neerl* 3 322
- SCHALM L (1951) Critische beschouwingen over de waarde van het moderne dieet bij ziekten van het leverparenchym *Voeding* 12 401
- SCHALM L (1952) Het regeneratievermogen en de reservecapaciteit van de lever en het mogelijk nut daarvan voor de chirurgie der galwegen *Acta Clin Belg* 7 156
- SCHWELLING J W (1934) Een bijzonder geval van aangeboren multiple gezwellen in de lever (hamartomen) bij een kind van vier maanden *Ned Tijdschr Geneesk* 3 3566
- SCHMIDT H and GUTTMAN (1954) Röntgenuntersuchungen über die Verzweigung der grossen intrahepatischen Gallenwege *Anat An.* 100 277
- SCHVEDORF, J G and ORR T G (1942) The effect of distention of the small intestine, anoxemia and oxygen therapy upon the flow of bile and urine in the dog. Relationship to the hepato-renal syndrome *Surg Gyn and Obst* 74 446
- SCHORNAGEL H E and STRAUB M (1954) Over het voorkomen van galblaascarcinoom. In *Vierde Jaarb 1 kankeronderz. en kankerbestrijding in Nederl* J H de Bussy, Amsterdam
- SCHROEDER W E (1906) The progress of liverhaemostasis. Reports of cases *Surg Gyn and Obst* 2 52
- SCHROEDER VAN DER KOLK I C DEFFLMAN
- SCOTT J V (1951) Use of absorbable gelatine sponge and primary suture in traumatic rupture of the liver *Amer J Surg* 81 371
- SECALL H V (1953) An experimental investigation of the blood and bile channels of the liver *Surg Gyn and Obst* 37 152
- SÉNÉQUE J and ALROUSSEAU R (1950) Des possibilités de l'hépatectomie dans les traitements des tumeurs du foie *J de Chir* 66 22
- SÉNÉQUE J, ROUX M, CHATELAIN CH and HUCLEFARD P (1952) Sur 1 cas d'hépatectomie typique gauche réglée pour kystes hydatiques

- multiples de la partie gauche du foie *Mém de l'Acad de Chir* 78 728
- SENEVIRATNI R D (1919) Physiological and pathological responses of the bloodvessels of the liver *Quart J Exp Physiol & Cognate Med Sc* 33 77
- ✓SERRA H (1901) Contribution à l'étude de la circulation du sang porte dans le foie et des localisations lobaires hépatiques *J de Méd Bordeaux* 31 271 291 312
- SHALLON I A and WAGNER (Jr) E B (1947) Primary fibrosarcoma of the liver *Ann Surg* 125 439
- SHEPHERD H I (1930) An embryonic tumour of the liver containing striated muscle *J Path Bact* 33 251
- SHIFFIELD W (1947) Cholecystectomy and partial hepatectomy for carcinoma of the gall bladder with local liver extension *Surgery* 22 48
- SHIDEMAN E I, KELLY A R, IVE I F, LOWELL A T and ADAMS B J (1949) The role of the liver in detoxication of thiopental (pentothal) by man *Anesthesiol* 10 421
- SHORR I (1950) Recent findings concerning the role of the liver and kidney in circulatory homeostasis. In *Liver injury Tr 8th Conf April Josiah Macy (Jr) Found* p 60
- SHORR I, ZWEIFACH B W and FURCHCOTT R F (1945) On the occurrences sites and modes of origin and destruction of principles affecting compensatory vascular mechanisms in experimental shock *Science* 102 489
- SILLMACKER (Jr) H B (1911) Hemangioma of the liver *Surgery* 11 209
- SIMMONDS M (1884) Die knotige Hyperplasie und das Adenom der Leber *Deutsch Arch Klin Med* 34 388
- SKIFOSOVSK (1896) *Le MARIE*
- SNAPPER I and SCHAEFER L F (1951) Fxanguino-transfusion in hepato-renal syndrome *Ann Int Med* 34 692
- SNYDER J M (1946) Surgical management of war wounds of the abdomen *Amer J Surg* 331
- SNYJERS I P and STRAUB M (1922) Over het primair levercarcinoom in de tropen (naar aanleiding van 57 waargenomen gevallen) *Geneesk Tijdschr Ned Ind* 62 3
- SOLOVON H A and KREPS S I (1950) Twenty six years of survival following carcinoma of sigmoid with prolonged liver metastasis *J Amer Med Ass* 144 221
- SPICELIUS A (1622) *De corporis humani fabrica* Frankfurt I c MEYER F
- STATE D, RAUCH R and MULLER J J (1951) The experimental production of the hepato-renal syndrome in dogs *Postgrad Med Surg Surg Forum Amer Coll Surg* p 171
- STEINER M M (1938) Primary carcinoma of the liver in childhood report of two cases with a critical review of the literature *Amer J Childh Dis* 55 807
- STEPHANSON G W (1932) Experimental pathology of the liver *Arch Path* 14 484

- STEWART M J (1931) Precancerous lesions of the alimentary tract *Lancet* II 563 and 617
- STIEDA L (1900) Anatomisch-archaeologische Studien Ueber die ältesten bildlichen Darstellungen der Säugetierleber *Anat Hefte* I 15/16 673
- STOUT A P (1943) Haemangioendothelioma a tumour of blood vessels featuring vascular endothelial cells *Ann Surg* 118 443
- STREETER J L (1945) Developmental horizons in human embryos *Contribution to embryology* 31 198 and 206
- SUMA F N and WILLIAMS R D (1951) Carcinoma of the gall bladder An analysis of cases *Ohio St Med J* 47 927
- SUMMERS J E (1952) Abdominal incisions I present status in the United States *Amer J Surg* 84 177
- TERRIER F and AUVRAY M (1907) *Chirurgie du foie et des voies biliaires Echinococcose hydatique commune Kystes alvéolaires Suppurations hépatiques Abscess tuberculeux intra-hépatique Absces de l'actinomycose* Félix Alcan Paris
- THOMAS L J and ZIMMERMAN H J (1952) The pattern of abnormality of liver function in metastatic carcinoma *J Lab Clin Med* 39 882
- THOMPSON J E (1899) The surgical treatment of neoplasms of the liver *Ann Surg* 30 284
- THOMPSON P (1908) A note on the development of the septum transversum and the liver *J Anat Physiol* 42 170
- THOMPSON R B (1947) Thrombosis of the hepatic veins the Budd Chiari syndrome *Arch Int Med* 80 602
- THOMSON A (1899) The morphological significance of certain fissures in the human liver *J Anat Physiol* 33 22
- THOREK M (1947) Partial hepatectomy in carcinoma of the gall bladder *J Int Coll Surg* 10 369
- TIEGEL M (1911) Zur Verhütung der Luftaspiration bei Venenverletzungen *Zentralbl f Chir* 38 1019
- TINKER M B (1935) Liver resection *Ann Surg* 102 728
- TINKER M B and TINKER (Jr) M B (1939) Resection of the liver *J Amer Med Ass* 112 2006
- TORI G (1953) Hepatic venography in man *Acta Radiol* 39 89
- TURNER G C (1923) Case in which adenoma weighing 2 lb 3 ozs was successfully removed from the liver with remarks on the subject of partial hepatectomy *Proc Roy Soc Med (Sect Surg)* 16 43
- VADHIM J L, GRAY H K and DOHERTY M B (1944) Carcinoma of the gall bladder A clinical and pathological study *Amer J Surg* 63 173
- VARADI S (1937) *Le Warvi*
- VARS H M and GLUD F V (1947) Effect of dietary protein upon the regeneration of liver protein in the rat *Amer J Physiol* 151 399
- VESALIUS V (1543) *De humani corporis fabrica* Ex off J Oporini Basilae
- VIRCHOW R (1858) Zur Diagnose und Prognose des Karzinoms *Arch Path Anat Physiol* 111 1

- WAHREN H and VINCKEN I (1931) Oblique splitting of the sternum in high gastric surgery *Acta Chir Scand* 107 15
- WAMM K C (1933) The blood supply of the normal liver *Proc Staff meetings Mayo Clin* 28 218
- WALKER J M and WYNN-PARRY C II (1939) The effect of hepatectomy on the action of certain anesthetics in rats *Brit J Pharm Chemother* 4 93
- WALLACE R H (1931) Resection of the liver for hepatoma *Arch Surg* 43 14
- WALLACE I c HAMILTON BAILEY
- WALLFUS (1630) *De motu chyli et sanguinis* Ad T Bartholinum epistola secunda
- WALTERS W and SNELL V M (1930) *Diseases of the gall bladder and bile ducts* W B Saunders Philadelphia London
- WALTHER H F (1938) *Arbeitsmetastasen* Benno Schwabe & Co Basel
- WANCENSTEIN O H (1935) Primary resection (closed anastomosis) of rectal ampulla for malignancy with preservation of sphincter function *Surg Gyn and Obst* 81 1
- WARVI W N (1944) Primary neoplasms of the liver *Arch Pathol* 37 367
- WARVI W N (1945) Primary tumours of the liver *Surg Gyn and Obst* 80 643
- WASSINK W F (1915) Halfzijdigheid van leverontaarding als gevolg van de verdeling van het bloed der poortader *Ned Tijdschr v Geneesk* 59 2145
- WEBBER I M (1927) Grades of malignancy of primary cancer of the gall bladder *Surg Gyn and Obst* 44 756
- WESE M S DE and LEWIS (Jr) C (1931) Partial hepatectomy in the dog An experimental study *Surgery* 30 642
- WEISS S (1944) *Clinical lectures on the gall bladder and bile ducts* The Year Book Publ Inc Chicago
- WELCH C F and GIDDINGS W P (1930) Abdominal trauma A clinical study of 200 consecutive cases from the Massachusetts General Hospital *Amer J Surg* 79 257
- WENDEL W (1911) Beiträge zur Chirurgie der Leber *Arch Klin Chir* 95 887
- WENDEL W (1920) Ueber Leberlappenresektion *Arch Klin Chir* 114 982
- WIEBERDINK J (1930) Over metastasering van kanker *Diss Groningen* Van Gorcum Assen
- WIGGERS C J (1945) The failure of transfusion in irreversible haemorrhagic shock *Amer J Physiol* 144 91
- WIGGERS C J, OPDYKE D F and JOHNSON J R (1946) Portal pressures gradients under experimental conditions including haemorrhagic shock *Amer J Physiol* 146 197
- WILES C F, SCHENK W G and LINDENBERG J (1932) Influence of

- hepatic artery ligation on regeneration of liver tissue in the rat *Arch Surg* 64 783
- WILLER H (1935) Ueber Lebergewebsembolie und das gewöhnliche Verhalten aus dem Verbannde gelösten Leberstücke im Organismus *Zentralbl Allg Path Path Anat* 62 209
- WILLIS R A (1943) Carcinoma arising in congenital cysts of the liver *J Path Bact* 55 492
- WILLIS R A (1948) *The pathology of tumours* Butterworth London
- WOLBACH S B and SAKAI T (1909) A new anaerobic spore bearing bacterium commonly present in the liver of healthy dogs and believed to be responsible for many changes attributed to aseptic autolysis of liver tissue *J Med Res* 21 267
- WOLFF W J (1950) Disrupture of abdominal wounds *Ann Surg* 131 534
- WOODHATT R T, SANBURN W D and WILDER R M (1915) Prolonged accurately timed intravenous injections of sugar A preliminary report *J Amer Med Ass* 65 2067
- WRIGHT T, PRIGOTA and HILL L M (1947) Traumatic rupture of the liver without penetrating wounds A study of 37 cases *Arch Surg* 54 613
- YEOMANS F C (1909) Primary carcinoma of the liver with report of a patient who remained well two years after operation *J Amer Med Ass* 52 1741
- ZONDAANEL E (1923) Abnorme Spaltbildung der Leber mit Distopie der Gallenblase *Zentralbl Allg Path Path Anat* 90 29

AUTHOR INDEX

- Atrancet 225 231
 Aiga 140
 Allrecht 128
 Albright 187
 Alford 203
 Anderson 131
 Anschutz 153 160 181
 Anson Bo
 Arden 143
 Auring 225 232
 Auroseau 120 125
 Auvray 149

 Barclay 43
 Barenzay von 131
 Bariaill 208
 Baron 151 209
 Baum 9
 Bay 156 160 173 193
 Benson 128
 Benz 197
 Bergmann von 120
 Berman 121
 Bernard 114
 Bchat 114
 Biering 135
 Blalock 140
 Boljarsky 149 151
 Boliman 203
 Bonne 121
 Bourne 206 207
 Boyce 148 216 217
 Breedis 232
 Broders 138
 Brunschwig 127 133 134 201
 Buchner 201
 Burckhardt von 149
 Burnett 147 149
 Burton Brown 143
 Burton Opitz 208
 Busing 146

 Campi 225 234

 Canthe B 12 113 158
 Carter B N 181
 Carter R F 224
 Castle 128
 Cathala 121
 Charnock Bradley 42
 Child 210
 Choronshtzky 42
 Clement 153
 Clute 187
 Cohnheim 69 208
 Cooper 137
 Copher 66 132
 Couinaud 12 21 31 34 37 38 64
 111 159 166 169 170 173 174
 178 181
 Counsellor 12
 Cousins 153
 Crump 135
 Cruveilhier 70
 Cuvier 6 9

 Dansie 122
 Davis 201
 De Bakey 147 148
 De Burlet 7 8 29 41 45 108 113
 115
 Deelman 120
 De Graef 219
 De Josselin de Jong 213
 Delorme 212
 Denton 201
 Descomps 10
 Devc 59 66
 De Vos 203
 Dick 66 132
 Dock 203
 Douglass 65 132
 Dubourg 142
 Duvernoy 6 8 9

 Edler von 141 149
 Eggel 122

Flas 1^a 29 53 99 101 112
 Ellenberger 9 112
 Flehjem 204
 Fpstein 147
 Escher 15^a
 Estrada 213
 Eustachius 4
 Ewing 120 129 130 136

Fabricius Hildanus 149
 Fairchild 150
 Ferrein 80
 Ferrier 149
 Ficarra 217
 Fine 212
 Finsterer 135 147
 Fletcher 133
 Flower II
 Flowers 36 113
 Forbes 204
 Frazer 209
 Frerichs 135
 Friedman 211
 Furtwaengler 148

Galenus 3
 Gans 21 93 193
 Garlock 136 139 186
 Garré 152 153
 Gerding 129
 Gershbein 112
 Gilbert 121
 Gilfillan 65 132
 Girard 8
 Glénard 66
 Glisson 8 11 12 59 92
 Gluck 199
 Glynn 203
 Golligher 133
 Gordon Taylor 142
 Graham F A 134 136
 Gray 137
 Cross 128
 Crover 210
 Curd 201
 Gurlt 9

Haberer von 69 10 208 209
 Haller von 4 30 43 47 50 68 69
 108 112 115
 Hamilton Baily 145
 Hammer 197
 Hanot 121

Hanseman von 138
 Hartroft 209
 Hasting James 130
 Healey 1^a 21 31 37 38 64 69 10
 73 159 166, 169 170
 Helwig 148 116
 Hendrick 130
 Henle 68 69
 Herben 125
 Hertwig 40
 Heyd 216
 Hildebrand von 16 120
 Himsworth 203
 Hippel 128
 Hus 43
 Hjortsjo 12 21 34 31 38
 Hoagland 204
 Holman 187
 Hromada 80
 Humphreys 186
 Hyden 134
 Hyrtl 12 68 69 79 91 220

Ihle 40
 Illingworth 134
 Ivy 201

Jankelson 135
 Johnston 80
 Jones 134
 Jurgens 214

Kaufmann 1 11 131 214
 Kay, 129
 Keen 156 157
 Kehr 91 141
 Kiernan 10 11 80 114
 Kimmel 211
 Kocher 153
 Kreps 133
 Kusnetzoff 151

Ladd 128
 Laloubne 10
 Lam 131
 Langenbuch 151 157
 Lee 128
 Lefèvre 132
 Leger 225 232
 Leys 9
 Leberkühn 220
 Lebow 222
 Luck 143 145 146

Locsho 200
 Lorl 07
 Lortat Jacob 132 133 186 213
 Lowe 209
 Luschka, G

Madding 129
 Mall 42 45 47 101 107 114
 Mann 200 201
 Markowitz, O 209 215
 Martens 16 158 220
 Martin 144 157
 Martina 154
 Mason F C 148 215
 Masse 142
 Mayer 149
 Mc Dill 153
 Mc Donald 148
 Mc Fetridge 216
 Mc Indoe 12
 Mc Rae 128
 Mekie 205 207
 Mermans 66 132
 Meyer 113
 Mickulitz 153
 Middlemiss 205 232
 Mikal 143 144 147 149
 Morrison 203
 Murray 211

Narath 208
 Nasse 00
 Newman 04
 Nicholls 122
 Noegenrath 122
 Noetzel 215
 Norman 91

Neill 144
 Opie 203
 Orr 213 216
 Otis 149
 Owen 9

Pareira 205
 Patton 128
 Payr 138 14 154
 Peck 210
 Pecquet 10 219
 Penberthy 128
 Pensky 154
 Pernet 160
 Petty 12 9 53 99 101

Philipp 128
 Phillips 126 131
 Ionfick 199 200
 Probststein 206

Quattlebaum 133 160

Rafsky 204
 Raginsky 207
 Ransohoff 153
 Raport 80
 Rapp 111
 Rappaport 114
 Ravdin 203 204 13
 Raven 158 184
 Reckhinghausen von 214
 Rex 6 12 21 34 34 59 66 79 93 95
 Ribbert 121
 Riddler 129
 Riedel 137 139 140
 Rio Branco 47 68 69 186
 Robert 13 159 186
 Rolleston 6 8 9
 Romieu 209
 Roth 128
 Rudbeck 219
 Rudstrom 147
 Ruge G 6 7 11 29 30 31 34 38
 95 108 112 113 115
 Ruge E 80
 Ruysch 2 0

Sabourin 114
 Sak 08
 Saunburg 136 139
 Saltz 200
 Saypol 224
 Schalm, 16 156 199 00 201 03
 Schmelling 1 9
 Schmorl 214
 Schmiedorf 13
 Schornagel 134 136
 Schroeder 149 153
 Schroeder van der Kolk 131
 Schroy 111
 Scott 153 205
 Segall 16 69 70 177 209 0
 Seneque 1 0 125 158 160
 Seneviratne 211
 Sezege 66 132 133 215
 Setterlee 131
 Sharpe 137
 Sheehan 1 11

Sheinfeld 142
 Shideman 206
 Shumacker 179
 Simmonds 120
 Skifosovsk 128
 Smith 203
 Snell 135
 Solomon 133
 Spiegelus 4 6
 Steiner 122
 Stewart 135 136
 Stueda 8
 Straub 134 136
 Suma 140
 Summers 184
 Sylvius 6
 Talbert 129
 Tannhauser 203
 Thomas 224
 Thompson P 40
 Thompson R B 211
 Thorek 142
 Tiegel 204
 Tinker 152
 Tori 225 23
 Vadheim 135 136 138 139
 Valerius 10
 van Horne 219
 Varadi 120
 Vars 201 213

AUTHOR INDEX

Vesalius 3 4
 Vikgren 187
 Virchow 130
 Wahren 184 187
 Walcus 10
 Walker 206
 Wallace 143
 Walters 135
 Walther 131 132 134 135
 Wangensteen 133 156
 Warvi 119 120 121 122 127 1 8 179
 Wasunk 13 215
 Webber 138
 Wendel 120 127 150 151 153 151
 157 158
 Whipple 201
 Wieberdink 132 133
 Wiggers 212
 Wiles 01
 Willer 214
 Williams 140
 Willis 127 128 130 133
 Wolbach 208
 Woodjatt 203
 Wright 143 144 145 147 149
 Wynn Parry 206
 Yamagna 128
 Young 232
 Zimmerman 224
 Zwammerdam 213

SUBJECT INDEX

- Abcess formation frequency 215
- Abdominal distension 113
- Adenoma as carcinoma 121
 - hepato-cellular 119 120 121
 - malignant 121
- Adenomas of intrahepatic bile ducts 119
 - liver cell 119 120 121
 - solitary 119
- Adrenal gland right 189
- Air embolism, 204
- Anaesthesia 205
- Anastomoses arterial between branches of hepatic artery and phrenic artery 69 70
 - — between right and left hepatic artery 16 17
 - — in fossa umbilicalis 69
 - — subcapsular arterial 79
- Anastomoses between bile duct systems 16
 - of the portal vein beds 9
- Anorexia 102
- Anoxia, prevention of 107
- Anti-coagulant therapy 213
- Arteriae hepaticae accessoriae 68
- Arteria (lobi) dextri 63
- Arteries variations in intrahepatic 87-91
- Artery cystic 51
 - hepatic 51
 - — aberrant 68
 - — and oxygenation of liver 108
 - — course and branches 60 63
 - — development 47
 - — site of division 70
 - — extrahepatic variations 67 68 69
 - — surgical details 177 178
 - — variations in number 68 69
 - left hepatic division 72-74
 - — situation 53
 - left paramedian 178
- Artery right hepatic 197
 - right paramedian 63
 - — ramifications 74-9
- Ascites 211 213
- Atelectasis 213
- Augmenta hepatis 1
- Auriga 2
- Bile duct common 51 63
- Bile ducts development 41
 - pattern of division 63
 - extrahepatic 80
 - intrahepatic variations 87-91
 - ramifications 79 80
 - surgical details 179 180
- B & A terminology 6
- Bronchopneumonia 213
- Bud hepatic pars cystica 41
 - — pars hepatica 41
- Cancer massif 121
- Capsule hepatic 10 49
- Carbohydrates 203
- Carcinoma cholangio-cellular 119 127
 - hepato-cellular 111-127
 - of the massive type 122
 - papillar 137
 - primary massive encapsulated 121
- Carcinoma of gall bladder 195 33
 - 34
 - cholecystectomy and 135 136
 - cholelithiasis and 135
 - incidence 131 135
 - liver and 138 139
 - mortality 134
 - perforation 137
 - sites of predilection 136
 - types 136 137
- Carcinomas liver cell 119 121-127
- Cardiac catheterization 105 232
- Caudal hilar approach 174
- Cholangiography operative 124
 - transabdominal 124

- Cholangio-hepatomas 119 128 179
 Cholangiomas 119 127
 Cholerrhagia 114
 Choristomas 178
 Cirrhosis of the liver 119
 Clamps 153
 Colliculus caudatus 6
 — papillaris 6
 Culter 2
 Cyclopropane 207

 Detoxication process of 203 206
 Diaphragm 189
 Diets 201
 Diodrast 232 234
 Dissemination extra and intrahepatic 126
 Dog's liver 4 7
 — differences with human liver 109
 Drains 211
 Duct see also Ductus
 — cystic 63
 — dextro-caudal 63
 — dextro-cranial 63
 — left hepatic 53 63 179
 — main hepatic 63
 — right hepatic 63 8 179
 — right lobar 63
 — right paramedian 180
 Ducts aberrant 80
 — common hepatic 179
 — hepatic variations at site of discharge 81-87
 — of Luschka 80
 Ductus see also Duct
 — caudalis lobi sinistri 63
 — choledochus 63
 — cranialis lobi sinistri 63
 — hepato-cardiacus 43
 — venosus (Arantii) 21 55

 Elastic ligature compression 153
 Embolism liver cell after hepatic trauma 214
 Endarteries of Cohnheim 108

 Fan dorsal 59 93 166
 — ventral 59
 Fibræ 2
 Fissura dorsalis 36 93 179
 — interlobaris dextra 31 31 166 182
 — — general position of 21
 — — site of 34
 — — surgery on 197

 Fissura interlobaris dextra variations 167-170
 — — variations of the intrahepatic course of 21
 — interlobaris sinistra 29 30
 — — site of 21 160
 — — surgery on 197
 — intermedia 38 166
 — intersegmentalis lobi sinistri 38
 — media 13-15 16 29 34 165 188
 — media histological examination 19
 — sagittalis dextra 7
 — sagittalis sinistra 7
 Fissures in the foetal liver 45
 — sagittal interlobar 159
 Focus 2
 Folic acid 204
 Fovea cystica 7 12 30 31
 — — division of 21
 — — fissure in 34
 — — position of 134
 — ductus venosi (Arantii) 4
 — sagittalis dextra 4 29
 — sagittalis sinistra 4 30
 — umbilicalis 30 67
 — venae cavae 4 30 92
 — venae umbilicalis 4
 — vesicae felleae 4
 Function tests liver 224
 Functional reserve capacity 199

 Gall bladder capillaries of 16
 — development of 21
 Gas producing bacilli 209
 Glissonian capsule 10 11 49 197
 — pedicles 54 63 165 166 173 197
 — structures topography 51
 — system 49-63

 Haemangioma cavernous 129
 Haematoma 144
 Hamartomas 128
 Hamartomy 128
 Haruspices 8
 Haruspicina 1
 Hemihepatectomy 11, 160 173 180 205
 — left 158 184
 — right 180 189
 Hepar dexter and sinister 34
 Hepatic failure 200 216
 Hepatic function depression of 107
 Hepato-blastomas 128

- Hepato-lobectomy left 115 193 194
 — middle 115 195 197
 — right 115 197
 Hepatomas 119 127
 Hilum 49
 — development 47
 — enlargement of the 174
 — variations in the 67
 Hyperglycaemia 205
 Hyperplasia nodular 119 120
 Hypoproteinaemia 202 203

 Incision abdomino-thoracic 186
 — sternum-splitting 184
 — subcostal 187
 — types 184
 Incisura dextra 50
 — umbilicalis 7 21
 Infections 215
 Injection-corrosion technique 219-223
 Injuries percutaneous 141 145
 — subcapsular 141 145
 — subcutaneous 143 144 146
 — symptomatology of 147 148
 — transcapsular 144 145
 — traumatic of the liver 143
 Injury and abscess formation 145
 — and cyst formation 145
 Intrahepatic systems spatial inter
 relationship in dog's liver 108
 — spatial inter relationship in human
 liver 108
 Intubation of the patient 205

 Kehr's sign 147

 Laparotomy exploratory 183
 Ligamentum coronarium 49 93
 — falciforme 49
 — hepato-diaphragmaticum 189
 — hepato-renal 189
 — triangulare 49 93 189
 — venae cavae 93
 — venosum (Arantii) 6
 Lipotropic agents 203
 Liver modes of subdivision 112-116
 — external subdivision 1-8
 — internal subdivision 8
 Lobar areas demarcation of the 29
 Lobe see also Lobus
 — central suspensory 6
 — left 165
 — — resection of 184
 Lobe left site of 16
 — middle 166
 — dorsal paramedian 159
 — — paramedian 197
 — — branches of 176
 — right paramedian 38 197
 — ventral paramedian 159
 — quadrate site of 16
 — right 171
 Lobectomy middle 182
 Lobe principale moyen 6
 Lobes paramedian 107
 Lobules primary 42
 — primitive 43
 Lobuli paramediales dorsales (cau
 dati) 47
 Lobus see also Lobe
 — anterior ■
 — caudatus 6 36 38
 — caudatus (Spigeli) 6
 — centralis ■
 — centralis sinister ■
 — descendens 8
 — dexter 4 34 36
 — — (BNA terminology) 7
 — — subdivision of 39
 — dextro-venalis 8
 — dorsalis 8
 — exiguus 6
 — lateralis dexter ■
 — lateralis sinister ■
 — medius 4
 — minimus 6
 — omentalis 6
 — paramedialis dorsalis dexter 36 39
 — — dorsalis sinister 36 39
 — — ventralis dexter 34 39
 — — ventralis sinister 34 39
 — posterior 6
 — praeportalis 6 8
 — quadrangularis 6
 — quadratus 6 38
 — quadratus (BNA terminology) 7
 — sinister 4 34
 — — (BNA terminology) 7
 — — subdivision of 39
 — venae cavae ■ 8

 Mammalian livers development 42
 Mensa 2
 Mesentery caval 93
 Metastases incidence in liver 131
 — localisation in liver 133

- Metastasis isolated 133
 Microorganisms : spore bearing and
 aerobic 208 212
 Necrosis hepatic 208
 Negative intrathoracic pressure 205
 — nitrogen balance 207
 Niacinamide 204
 Nicotinamide 204
 Nomenclature for internal division 34
 — old 6
 Omentum lesser 49
 Paralytic ileus 213
 Parenchyma development of liver 40
 Pars dextra hepatis 36 38
 — sinistra hepatis 38
 Pedicles Glissonian 54 63 165 166
 173 197
 — lobar 63
 — segmental 63
 — vascular retraction and compres-
 sion of 207
 Penacula 2
 Pentothal 206
 Penulae 2
 Phlebography, operative portal 225
 — transabdominal portal 225
 Pig's liver 4 7 113
 Plane avascular 158
 Plane of resection fissures as 159
 Porta hepatis 4
 Protein deficiency 202
 Proteins 203
 Prothrombin concentration 207
 Pulmonary complications 213
 Radiocutting 152
 Recessus umbilicalis of Rex 21
 Resection 150
 — of solitary metastases in the liver
 133
 — of the pars dextra hepatis 115 133
 167 195 197
 Retractor eyelid 174
 — Finocchio 186
 Saccus 54
 Segmentectomies 201
 Segments division of the liver lobes
 into 36
 Segmentum caudale lobi dextri 38
 Segmentum craniale lobi dextri 38
 — cranio-dorsale lobi sinistri 38 57
 — ventro-caudale lobi sinistri 38 57
 Septum transversum 40
 Sheep's liver model of 1 2
 Shock effect on condition of liver 211
 — irreversible 212
 Signet ring cells 137
 Sinusoids 40 41
 Sinus venosus 47
 Splenomegaly 213
 State of organ preoperative 20
 Sulcus transversus 4 50 53
 Surgery on dogs 171
 Sutures 154
 Syndrome Budd Chiari 210
 — hepatorenal 148 213
 Tamponade 150 153
 Terato-carcinomas 1 8
 Teratomas 128
 Thermocautery 152
 Thiamin 204
 Thiopental 206
 Treatment conservative in hepatic
 trauma 149
 Truncus communis 95 97
 — dexter venae portae 51 17 67
 — sinister venae portae 51
 — velleo umbilicalis 42
 Tuber 6
 Tumour growths secondary types
 232 233
 — index 138
 Tumours benign 118
 — liver embryonic 128
 — grading of 138
 — hepatic blood supply in 232
 — malignant 118
 — metastatic in the liver 130
 — primary hepatic 119 130
 Unit structures morphological and
 functional 114
 Vcm see also Vena
 — portal 12
 — — development 43
 — — extrahepatic variations 61
 — — occlusion of 209
 — — stream lined effect of 11 and 61
 132
 — — surgery 1 177

SUBJECT INDEX

- Ven portal thrombosis 213
 - left portal 21
 - right portal 138
- Ven system the hepatic 12 92 107
- Ven vitelline 107
- Veins hepatic obstruction of 210
 - pressure in the 20
 - shunts between 101
 - major hepatic 188
 - major hepatic interlobaric 93
 - umbilical 42
 - vitelline 41
- Vena cavae
 - caudalis lobi sinistra 67
 - cava inferior 12 92
 - coronaria sinistra 64
 - cranialis lobi sinistra 37
 - variation of the 17
 - dextra 59 60
 - dextro-caudalis 60
 - dextro-cranialis 60
 - dextro-marginalis 60
 - hepatica dextra 93 99 161
 - — dialis 99
 - — dorso-lateralis dextra 99
 - — media 97 97 161 182 188 197
 - — sinistra 12 93 95 97
- Vena hepatis 63
- mesenterica magna superior 63
- mesenterica parva sive inferior 1
- parajuxta 60
- ramification 167 169 170
- paramedialis dextra 57 67 176 181
- sinistra caudalis superior 53
- sinistra cranialis 57 176
- Vena caudatae sinistrae 51
- hepaticae breves 93 93 188 189
- caudatae 99
- paramediales sinistrae caudales 57
- Venogram hepatic 232
- Venous collateral circulation 66
- Venous system hepatic development 41
- Venula hepatica cranialis dextra 99
 - cranialis sinistra 97
 - interlobaris sinistra 97
 - medialis sinistra 97
- Venules hepatic 193
- Vitamins 94
- Wangensteen suction 213
- X-ray treatment 126

